The Role of the Prefrontal Cortex in the Expression of Impulsive- and Premeditated-Aggression

by

Sarah Haberle BA (Hons)

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School of Psychology
University of Tasmania
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Abstract

The notion that there is a relationship between frontal lobe damage and aggressive behaviour has been recognised in the clinical literature for over 50 years. However, although there is evidence for an association between general brain dysfunction and aggression, there is little evidence pertaining to subclinical impairment and the propensity for aggressive behaviour. Further to this, given the functionally heterogeneity of the prefrontal cortex, it is vital to delineate the specific roles of the dorsolateral, orbitofrontal and medial aspects of the prefrontal cortex in the expression of aggression.

Two forms of aggression are distinguished: reactive, impulsive-aggression and goal-directed premeditated aggression. While impulsive-aggression is typically described as an emotionally-charged aggressive response characterised by a lack of control, premeditated aggression is considered to be a planned and controlled aggressive display that is instrumental in nature. The qualitative differences between these subtypes of aggression suggest distinct neuropsychological differences mediating the likelihood of their display.

The aim of this thesis was to clarify the role of the prefrontal cortex in subclinical impulsive-aggression and premeditated aggression. More specifically, possible executive functioning deficits mediated by the dorsolateral prefrontal cortex, and emotion recognition, impulsivity, and response reversal capabilities mediated by the orbitofrontal cortex were explored. Participants included university undergraduate students identified as having high levels of trait aggression, classified as either predominantly impulsive, or predominantly premeditated in nature.

Experiment 1 (n=85) explored possible executive deficits using a battery of

neuropsychological measures pertaining to dorsolateral functioning. It was found that impulsive-aggressive individuals performed significantly poorer on measures of cognitive flexibility, planning, problem-solving, and flexibility of verbal thought processes.

Experiment 2 (n=87) sought to identify possible deficits in interpretations of facial expressions of emotion and hostile attribution biases. Contrary to expectations, the results indicated that while impulsive- and premeditated-aggressive individuals do not incorrectly interpret emotional expressions, premeditated-aggressive individuals attributed greater levels of aggression to neutral faces.

Experiment 3 (n=87) investigated functions of the orbitofrontal cortex, namely impulsivity, response reversal, and decision-making capabilities. No differences between impulsive-aggressive and premeditated-aggressive individuals were found on any of these measures suggesting negligible involvement of the orbitofrontal cortex in subclinical aggression.

Overall, the results from this thesis suggest distinct neuropsychological processes in individuals who display predominantly impulsive-aggressive behaviour compared to those who display predominantly premeditated-aggression. While impulsive-aggression may result from executive dysfunction pertaining to the dorsolateral region of the prefrontal cortex, the display of premeditated aggression is related to functioning of the orbitofrontal cortex mediating the interpretation of aggression in others. Such findings have important implications not only in the understanding of the causal features of such behaviour, but also in the development and implementation of successful treatment strategies.

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Chapter 1

Overview of the Thesis

While the underpinnings of human aggression are clearly multifactorial, including political, socioeconomic, cultural, and psychological factors, it is also clear that some forms of aggression, either impulsive or premeditated in nature, have an underlying neurobiology that is only just beginning to be understood. In this research the neurobiology of aggression is addressed, specifically the role of the prefrontal cortex in the expression of both impulsive- and premeditated-aggression.

A significant body of evidence indicates that the likelihood of acting aggressively is related to the functional capacity of the frontal lobe. Using neuroimaging techniques, studies of violent offenders have consistently shown abnormalities in frontal lobe structures in individuals who have histories of violence (e.g., Raine, Lencz, Bihrle, LaCasse & Colletti, 2000; Raine et al., 1998). Additionally, lesion studies (e.g., Damasio, Grabowski, Frank, Galaburda & Damasio, 1994) and neuropsychological studies (e.g., Stanford, Greve & Gerstle, 1997) have provided evidence of the relationship between prefrontal impairment and the propensity for aggressive behaviour. Unfortunately, however, the above studies have placed little emphasis on the separable regions of the prefrontal cortex. This is despite the fact that neuropsychological data strongly suggesting that only medial and orbitofrontal regions of the prefrontal cortex are involved in mediating aggression, while dorsolateral prefrontal cortex has little role (Grafman et al., 1996). The prefrontal cortex is functionally and anatomically heterogeneous and thus the separable regions may be differentially involved in the expression of aggression. The present thesis thus attempts to delineate the specific roles of these subregions in impulsive- and premeditated-aggression.

Many researchers have suggested that the relationship between prefrontal abnormalities and likelihood of aggression is mediated by the failure to adaptively use executive functions (Giancola, 2000). As outlined in Chapter 5, executive functions is a broad term used to describe those abilities which allow an individual to respond to situations in a flexible manner, creating and adapting plans, and not being governed exclusively by external stimuli (Hoaken, Allaby, & Earle, 2007). Such abilities are presumed to be mediated predominantly by the dorsolateral region of the prefrontal cortex.

A further ability linked to the prefrontal cortex is the ability to correctly interpret emotional facial expressions. More specifically, patients with orbitofrontal cortex lesions are impaired in their ability to recognise facial expressions, particularly anger (Blair & Cipolotti, 2000; Hornak, Rolls & Wade, 1996). Neuroimaging studies support these findings, demonstrating activation in the orbitofrontal cortex by negative emotional expressions; in particular, anger, but also fear and disgust (Blair, Morris, Frith, Perrett & Dolan, 1999; Kesler-West et al., 2001). As described in Chapter 6, aberrations in the ability to identify facial expressions may result in the generation of inappropriate social responses, such as reacting aggressively to ambiguous social situations (Dodge, Laird, Lochman & Zelli, 2002). This hypothesis is based on Dodge (1986) who proposed that the accurate interpretation of social stimuli must be completed for prosocial behaviour to be manifested.

Individuals with lesions to the prefrontal cortex have also been shown to have deficits in inhibition, decision-making, and response reversal (Bechara, Damasio, Damasio & Anderson, 1994; Rolls, Hornak, Wade & McGrath, 1994). Such patients also display impairment in social functioning. The suggestion then follows that reappraisal will play an important role in social contexts in which one is required to

adapt to rapidly changing contexts (Happaney, Zelazo & Stuss, 2004). Furthermore, an inability to suppress previously rewarded responses due to inhibitory deficits, will in turn lead to inappropriate social responses.

In research on aggression, it is vital to distinguish between impulsive- and premeditated-aggression. Impulsive-aggression is more reactive in nature and displayed without a self-generated goal. Premeditated-aggression, in contrast, appears to occur without provocation, is proactive, and is seen as a means to gain a valued outcome. This heterogeneity between impulsive- and premeditated-aggression suggests distinct cognitive mechanisms responsible for their display.

Both the animal and human neuropsychological literature suggests that the prefrontal cortex is involved in the modulation of impulsive-aggression (Anderson, Bechara, Damasio, Tranel & Damasio, 1999; Gregg & Siegel, 2001). Certainly, damage to the medial frontal and orbitofrontal cortex is associated with increased risk for the display of impulsive-aggression in humans whether the lesion occurs in childhood (Anderson et al., 1999) or adulthood (Grafman et al., 1996). More specifically, individuals with orbitofrontal cortex lesions are typically described as disinhibited, socially inappropriate, impulsive, irresponsible, and as often misinterpreting others' moods (Rolls et al., 1994). In addition, there are considerable neuroimaging data assessing the neural functioning of patients with impulsive-aggression. These data have revealed reduced prefrontal functioning in patients presenting with impulsive-aggression (Søderstrom, Tullberg, Wikkelso, Ekholm & Forsman, 2000). Interestingly, this reduced prefrontal functioning is not observed in those with predominantly premeditated-aggression (Raine et al., 1998).

Accounts of premeditated-aggression often lie in the related construct of psychopathy. Psychopathic individuals, individuals who present with marked

premeditated-aggression, do not present with deficits on neuropsychological measures which pertain predominantly to the dorsolateral prefrontal cortex (Mitchell, Colledge, Leonard & Blair, 2002). Psychopathic individuals' high level of premeditated-aggression is thus completely unlike that of patients with orbitofrontal cortex lesions (Cornell et al., 1996). It is likely then that such individuals show elevated levels of premeditated-aggression because they have been reinforced, and not punished, for committing such behaviour in the past (Blair, 2004). Such aversive conditioning has been shown to be mediated by the amygdala. In support of this, an MRI study by Tiihonen et al. (2000) found a strong negative correlation between level of psychopathy and amygdala volume.

However, while there is clear evidence of amygdala involvement in premeditated-aggression through its role in aversive conditioning and instrumental learning, the orbitofrontal cortex may also be involved through its role in response reversal and extinction. That is, changing a response to a stimulus when the reinforcement contingencies change (Dias, Robbins & Roberts, 1996a). Moreover, the orbitofrontal cortex has been linked to decision-making when knowledge about potential positive and negative results is necessary to guide behavioural responding (Rogers et al., 1999b). On tasks such as the Intradimensional/Extradimensional (ID/ED) Set Shift Task which involves response reversal, adult psychopathic individuals show impairment (Mitchell et al., 2002). This suggests possible orbitofrontal dysfunction in individuals presenting with marked premeditated aggression.

Investigating prefrontal impairment in antisocial behaviour more broadly is problematic due to its heterogeneity and comorbidity with several disorders, including drug and alcohol abuse, Antisocial Personality Disorder (APD), pathological

gambling, schizophrenia, and bipolar disorders, all of which may or may not involve an aggressive component. Frontal lobe functions have been implicated in all of these comorbid conditions, however frontal lobe deficits have also been found in disorders not readily associated with antisocial behaviour, such as obsessive-compulsive disorder. The question thus lies in whether there is a single common component of these disorders given that studies on such populations have been inconsistent with regard to demonstrated neuropsychological deficits.

Through an investigation of prefrontal functioning in impulsive- and premeditated-aggressive individuals, the current study aimed to answer the following research questions:

1. Do impulsive-aggressive and premeditated-aggressive individuals perform differently on measures of executive functioning known to relate to dorsolateral prefrontal cortex functioning?
2. Do impulsive-aggressive and premeditated-aggressive individuals differ in their recognition of emotions in faces?
3. Do impulsive-aggressive and premeditated-aggressive individuals demonstrate a hostile attributional bias in their interpretation of neutral facial expressions or overestimate the level of aggression in aggressive faces?
4. Do impulsive-aggressive and premeditated-aggressive individuals present with deficits on measures of orbitofrontal cortex functioning, namely inhibition, decision-making, and response reversal?

Chapter 2

Impulsive- and Premeditated-Aggression – A Review of the Literature

2.1 Aggression

Aggression can be defined as "any behaviour directed toward another individual that is carried out with the proximate (immediate) intent to cause harm" (Bushman & Anderson, 2001, p. 274). The aforementioned definition of aggression encompasses a variety of behaviours, which can range from verbal to relational to physical (Crick & Grotpeter, 1995). For example, a widely used definition of aggression is behaviour deliberately aimed at harming people and/or objects (Dodge, 1991). In this definition, harm has implicitly been defined as hurting someone physically. However, other forms of harm, such as psychological harm (e.g., humiliating), and relational harm (e.g., malevolent gossip), are just as important when discussing the notion of aggression.

The aforementioned definition of aggression does not assume that all harmful behaviours are aggressive, rather, there are many instances in which harmful behaviours are prosocial. For example, the possible pain caused by a dentist to their patient is not aggressive because the proximate intent of the dentist is to help rather than hurt the individual. Similarly, both physical aggression in the context of self-defence and the selective use of verbal aggression by politicians, for example, are adaptive. Aggression, on the contrary, is problematic when it is a habitual behavioural pattern (Bushman & Anderson, 2001).

The notion of aggression has been used to refer to a wide variety of concepts and phenomena which has led to much confusion within the aggression literature due, in part, to interchangeable use with other terms (Caprara et al., 1985). The related

theoretical constructs of anger and hostility continue to be used in lieu of aggression and it is therefore necessary to elucidate the conceptual distinctions between these constructs by reviewing their operational definitions. As noted above, aggression refers to a behavioural process that includes the goal of inflicting harm to another individual or object. In contrast, anger is conceptualised as an emotional state that can vary in intensity, from mild annoyances to rage (Spielberger, Jacobs, Russell & Crane, 1983). Moreover, the experience of anger lacks a specific goal (Berkowitz, 1993) and is not necessary for aggression to occur. Unlike aggression or anger, hostility is an attitudinal or cognitive construct comprised of enduring cognitions that involve negative interpretations of the environment. As such, once hostile attitudes are verbally or physically expressed, they may be more appropriately labelled as aggression. Aggressive behaviour also needs to be distinguished from antisocial behaviour. Antisocial behaviour is defined as behaviour by which people are disadvantaged and basic forms and norms are violated (Merk, de Castro, Koops, & Matthys, 2005). Examples of such behaviour are lying, stealing and truancy. Aggressive behaviour, then, is a specific form of antisocial behaviour (Kempes, Matthys, de Vries & van Engeland, 2005).

Caprara and colleagues (Caprara et al., 1985) suggest that the use of such concepts should be restricted to the use of two primary concepts: aggressiveness and aggression. According to their view, aggressiveness refers to a personality characteristic, while aggression refers to the aggressive behaviours manifested.

2.2 Distinguishing between impulsive- and premeditated-aggression

Traditionally, the study of violence and aggression has recognised the importance of distinguishing between different types of such behaviour. Aggression

can be classified in a number of ways, for example, by the target of aggression (e.g., self-directed or directed towards another individual or object), mode of aggression (e.g., physical or verbal, direct or indirect), or cause of aggression (e.g., medical) (Siever, 2008). Although many individuals display more than one subtype of aggression (Barker, Tremblay, Nagin, Vitaro, & Lacourse, 2006), and correlations often exist among subtypes of aggression (Kempes et al., 2005), two distinct subtypes of aggressive behaviour consistently emerge; an affective or impulsive type, and a predatory or premeditated type. Characteristically, these two subtypes of aggression are distinguished by several features, but primarily by the amount of behavioural control exhibited during the incident.

Impulsive-aggression is described as a reactive or emotionally charged aggressive response characterised by a loss of behavioural control (Barratt, 1991; Raine et al., 1998). These aggressive acts are unplanned and spontaneous in nature and are either unprovoked or out of proportion to the provocation. Impulsive-aggression is usually accompanied by an agitated or irritated mood, poor modulation of physiological arousal and loss of behavioural control (Houston, Stanford, Villemarette-Pittman, Conklin & Helfritz, 2003). Interpersonal communication is often non-adaptive during the agitated state and information processing appears to be inefficient (Elliot, 1992). This subtype of aggression can result in sudden, heightened, or inappropriate aggressive responses, and probably accounts for most societal problems that are associated with aggression. Coccaro (1998) emphasises that while the impulsive-aggressive individual does not necessarily have the intention to cause harm either to themselves or to others, their behaviour nevertheless has the potential to do so. Perpetrators of impulsive-aggression often report regret after the act, however often lack the self control to refrain from committing such acts again

(Barratt, 1994). Barratt proposes that the personality traits of impulsiveness and anger/hostility are related to most impulsive-aggressive acts.

The theoretical roots of impulsive-aggression lie in the frustration-aggression model (Berkowitz, 1993). According to this theory, aggression is displayed as a consequence of frustration, actual or perceived threat, and heightened arousal in the form of anger. Aggression is displayed in reaction to aversive events with the subjective experiences of the individual central to the situation. The subjective experience of, for example, feeling threatened and not necessarily being threatened is a principal concept in this theory. Frustration may not immediately lead to aggression, but generate such emotions as anger, which can then augment the readiness to display aggression (Merk et al., 2005).

Premeditated-aggression, on the other hand, is considered a purposeful, controlled aggressive display that is usually instrumental in nature. These acts require forethought and planning and are generally executed with low autonomic arousal (Stanford, Houston, Villemarette-Pittman, & Greve, 2003b). Premeditated-aggressive acts are carried out with a high degree of behavioural control and are directed toward a goal, such as external reinforcers (e.g., money oriented) or intimidation (Dodge & Coie, 1987; Hubbard, Dodge, Cillessen, Coie, & Schwartz, 2001; Vitiello, Behar, Hunt, Stoff, & Ricciuti, 1990).

Premeditated-aggression can be understood by the principles of operant conditioning, where the probability of aggression is increased by prior history of subsequent reward or reinforcement (Dodge, 1991). An act of aggression occurs because of the expectancy of the reinforcement or reward that is to follow. From this model, one can see that the likelihood of committing an aggressive act may increase as a function of social reinforcement emanating from an environment where gangs are

present or where aggression is viewed in a more positive light (Kingsbury, Lambert, & Hendrickse, 1997). A deficit in the ability to experience or anticipate remorse or the aversive outcomes increases the risk of premeditated-aggression, which appears to be the case in aggressive individuals with psychopathy who have difficulty anticipating and experiencing negative feelings of remorse or guilt (Hare, 1999).

The utility of this impulsive-premeditated classification has been indicated in predictive validity studies (Barratt, Stanford, Felthous, & Kent, 1997a; Barratt, Stanford, Kent & Felthous, 1997b; Heilbrun, Heilbrun, & Heilbrun, 1978; Linnoila et al., 1983; Mungas, 1988). In a study involving male and female college students, Barratt and colleagues (Barratt, Stanford, Dowdy, Liebman, & Kent, 1999) found impulsive and premeditated acts of aggression are independent constructs, however, emphasised that these two subtypes of aggressive behaviour may also coexist to varying degrees in „normal' persons. It is rare in practice that the aggressive acts exhibited by an individual can be classified as entirely premeditated or entirely impulsive (Stanford et al., 2003b; Weinshenker & Siegel, 2002). For example, an individual who habitually loses his temper, exhibits an irritable mood and often responds out of proportion to the provoking stimulus might be characterised as displaying impulsive-aggression. However, it is possible that this individual may also demonstrate some incidences of premeditated-aggression. Conversely, an individual whose aggressive displays are usually planned and consciously executed may experience some instances in which he loses control of his behaviour. Thus the proposed dimensional as apposed to categorical classification scheme permits the behaviour to be characterised as predominantly premeditated or predominantly impulsive, allowing for the heterogeneity that naturally occurs within an individual.

Although there has been some criticism of the dichotomous method of characterising aggressive behaviour (see Bushman & Anderson, 2001; Parrott & Giancola, 2007), a dichotomous approach is supported by a number of important distinctions between individuals who express predominantly impulsive- or predominantly premeditated-aggression. More specifically, researchers have found that impulsive-aggressive individuals experience disruptions across a variety of domains including verbal ability and intelligence, physiological reactivity, biological function and treatment response (Barratt et al., 1997b; Coccaro, 1992). In contrast, individuals demonstrating premeditated-aggression tend to have more circumscribed disturbances on measures of personality (Barratt et al., 1997a; Stanford et al., 2003b).

2.2.1 Psychobiological evidence

The neurotransmitter that has received the most attention in regards to aggressive behaviour is serotonin. Serotonin facilitates prefrontal cortical regions, such as the orbitofrontal cortex and anterior cingulate cortex that are involved in modulating and often suppressing the emergence of aggressive behaviours (Siever, 2008). In both humans and animals, it appears that serotonin is primarily associated with impulsive-aggression in comparison to premeditated-aggression, and it appears that its effects may be receptor specific (Miczek, 1987; Shaikh, De Lanerolle, & Siegel, 1997). In humans, a relationship between decreased serotonergic function and impulsive-aggressive behaviour has consistently been demonstrated using a number of strategies, including central neurochemical measures (CSF 5-HIAA; Linnoila et al., 1983; Roy, Adinoff and Linnoila, 1988; Virkkunen, De Jong, Bartko, Goodwin, & Linnoila, 1989a), platelet binding (Kent et al., 1988), prolactin response (Fenfluramine; Coccaro et al., 1989), pharmacological treatment (sertraline; Kavoussi,

Liu & Coccaro, 1994; fluoxetine; Coccaro, Kavoussi & Hauger, 1997) and regional metabolic activity in response to serotonergic agonist (m-CPP; New et al., 2002). Barratt, Kent, Bryant and Felthous (1991) found that phenytoin reduces the frequency of aggressive acts. Replication studies have shown that phenytoin may reduce incidences of impulsive-aggression, but not premeditated-aggression (Barratt et al., 1997b; Barratt, Felthous, Kent, Liebman & Coates 2000). Such findings support the hypothesis that impulsive- and premeditated-aggression have different underlying biological substrates that respond differently to pharmacologic agents with specific modes of action.

2.2.2 Psychophysiological evidence

Psychophysiological techniques also provide a practical measure of neuropsychological functioning, however while a substantial literature exists on autonomic correlates of antisocial behaviour (Raine, 2002a, 2000b), there are a limited number of studies which have compared these measures in groups whose aggression was explicitly classified according to an impulsive-premeditated scheme. Pitts (1997) measured heart rate in aggressive children whose behaviour was classified as reactive (impulsive) or proactive (premeditated) in nature. Those characterised by reactive aggressive behaviour responded to a challenging task with increasing heart rate while those characterised by proactive aggression did not. Similarly, in a study of domestic batterers, heart rate activity was compared during marital interaction (Jacobson & Gottman, 1998). The batterers were divided into those whose heart rates lowered during the interaction and those whose heart rate increased. The men exhibiting decreasing heart rates were characterised as calm, calculated, antisocial and sadistic (premeditated). Those exhibiting increasing heart rates were

described as being more emotional, angry and unstable (impulsive). Finally, a more recent study of aggressive children indicated a significant increase in skin conductance reactivity in those rated high in reactive aggression during a laboratory-based measure of induced anger (Hubbard et al., 2002). Again, those individuals exhibiting primarily reactive or impulsive aggression responded differently autonomically than those deemed more proactive or premeditated.

Electroencephalography (EEG) abnormalities are also present among those who engage in impulsive-aggression. For example, Drake, Hietter and Pakalnis (1992) found a greater incidence of EEG slowing in a group of patients described as having episodic dyscontrol syndrome as compared to depressed patients and controls. Abnormalities in P1 amplitude have been reported in impulsive-aggressive college students (Houston & Stanford, 2001) and youths characterised by explosive aggressive behaviours (Bars, Heyrend, Simpson & Munger, 2001). In addition, adults classified as impulsive-aggressive exhibit decreased P1-N1-P2 latency (Houston & Stanford, 2001), reduced P3 amplitude (Barratt et al., 1997b; Gerstle, Mathias & Stanford 1998; Mathias & Stanford, 1999), increased P3 latency (Mathias & Stanford, 1999), and reduced amplitude and increased latency on the late positive potential, a purported measure of emotional processing (Conklin & Stanford, 2002). These differences reflect a number of sensory and information processing deficits specific to impulsive-aggression, as well as preliminary evidence for emotional processing impairment.

Volkow et al. (1995), using positron emission tomography (PET), found that psychiatric patients with a history of repetitive, purposeless violent behaviour showed significantly lower relative metabolic values in medial temporal and prefrontal cortices compared to normal controls. Similarly, Raine et al. (1998) reported that

affective (impulsive) murderers have significantly reduced prefrontal activation when compared to predatory (premeditated) murderers and controls.

The literature regarding premeditated-aggression though sparse is consistent. Individuals who engage in acts of premeditated-aggression show few differences from non-aggressive controls on psychophysiological measures, including P3 (Barratt et al., 1997b; Stanford et al., 2003b). Stanford et al. found that the P3 latency difference did approach significance (p = .06), suggesting a trend toward a longer P3 latency in the premeditated-aggressive group. Such prolonged P3 latency has been linked to increased attitudinal hostility (Bond & Surguy, 2000). Thus, the high levels of anger/hostility evidenced in the premeditated-aggressive group may have played a role in the latency trend observed in the sample.

2.2.3 Neuropsychological evidence

While small in number, neuropsychological studies comparing modes of aggression have established a correlation between increased impulsive-aggression and decreased executive functioning, while few deficits have been found in those who are premeditated in their aggressive behaviour (Houston et al., 2003). Dolan and Anderson (Dolan, Deakin, Roberts & Anderson, 2002) grouped male personality disordered offenders into high and low impulsive aggressors using the Belligerence Scale of the Special Hospital Assessment of Personality and Socialization (SHAPS: Blackburn, 1982). They found that both impulsivity and aggression were negatively related to executive function, and aggression was negatively related to memory function. Similarly, Giancola, Moss, Martin, Kirisci and Tarter (1996) found that problems in executive functioning were a predictor of reactive aggression in adolescent boys at risk for substance abuse. Most recently, Villemarette-Pittman,

Stanford and Greve (2002) found that verbal deficiencies varied according to executive demands of the task in a sample of impulsive-aggressive college students.

In the first study to compare premeditated-aggressive subjects with controls on a variety of neuropsychological tests, Stanford et al. (2003b) found no significant differences except for a single subscale of the Wisconsin Card Sorting Task (WCST), where the premeditated group exhibited greater failure to maintain set than controls. In contrast, there were pronounced differences on a range of personality measures, including impulsivity, verbal and physical aggression, anger, hostility, psychoticism and neuroticism. The authors concluded that the difference between the premeditated-aggressive group and controls was a result of an impulsive personality style rather than a significant cognitive deficit.

In summary, neuropsychological assessment has shown a clear link between impulsive-aggressive behaviour and problems in executive functioning, while few if any deficits have been demonstrated in premeditated-aggressive individuals.

2.2.4 Psychopathy

The concept of psychopathy has provided some utility in further distinguishing between impulsive- and premeditated-aggression. Psychopathy refers to a constellation of personality and behavioural characteristics marked by low baseline arousal, dishonesty, absence of remorse, empathy, and conscience, antisocial behaviour, and impersonal relationships (Hare, 2003). Interpersonally, they are often described as grandiose, arrogant, callous, superficial and manipulative (Hare, 1999).

The concept of psychopathy has been operationalised by the work of Hare (1991, 1999), and its assessment is now highly reliable and valid. There is a growing body of research to show that psychopathic criminals engage in more premeditated

and predatory violence than non-psychopathic criminals (Cornell et al., 1996; Serin, 1991). Williamson, Hare and Wong (1987) found that incarcerated psychopaths had higher rates (45.2%) than incarcerated non-psychopaths (14.6%) of committing their crime for material gain (i.e., proactive in nature), and that non-psychopaths had higher rates (31.7 vs. 2.4%) of emotional arousal leading to their offences (i.e., impulsive in nature). Likewise, Cornell et al. (1996) found premeditated-aggressive offenders could be distinguished from non-premeditated offenders by higher total psychopathy specifically concerning: pathological lying; manipulative actions; lack of empathy; parasitic lifestyle; irresponsibility; criminal versatility; and superficiality. The authors concluded that "the link between psychopathy and instrumental violence supports the distinction between instrumental and reactive violence, and raised the possibility that the presence of instrumental violence could be an associated characteristic of psychopathic offenders" (p. 790). Similarly, Woodworth and Porter (2002), in a study of 125 homicide offenders, found that the great majority of homicides committed by psychopaths were instrumental (i.e., premeditated), whereas only 48.4% of homicides committed by non-psychopaths were instrumental. Meloy (1988) theorised that a predisposition to engage in premeditated violence in psychopaths would be due to their low levels of autonomic arousal and reactivity, their disidentification with the victim, their emotional detachment and their lack of empathy.

Similar differences have been found in children. A growing body of evidence indicates that children with conduct problems represent a group that can be further divided into discrete subtypes based on the presence of callous and unemotional traits (Frick, O'Brien, Wootton & McBurnett, 1994; Frick & Ellis, 1999). Callous and unemotional traits are similar to psychopathic traits such as lack of guilt and empathy, superficial charm, and constricted emotion. In a sample of children with conduct

problems, Christian, Frick, Hill and Tyler (1997) found that a subgroup of children exhibiting symptoms of Oppositional Defiant Disorder (ODD) and Conduct Disorder (CD) and callous and unemotional traits differed from those without such traits in the number and variety of conduct problems. Pardini, Lochman and Frick (2003) found that the presence of callous and unemotional traits in adjudicated juveniles was associated with the use of aggression to obtain rewards and dominate (i.e., premeditated). Frick et al. (2003), in a sample of non-referred children, found that children demonstrating conduct problems and callous and unemotional traits were more likely to demonstrate high levels of proactive aggression than those without these traits, whose aggression was predominantly impulsive. It thus appears that it is the presence of callous and unemotional traits that distinguish this subgroup and its associated problems.

2.3 Importance of distinguishing between impulsive- and premeditated aggression

There is evidence in both children and adults that impulsive- and premeditated- aggression are distinguishable forms of aggressive behaviour with important clinical implications. For example, longitudinal studies in children and adolescents rated on reactive and proactive aggressive behaviour have shown that proactive, but not reactive, aggression predicts later delinquent behaviour (Vitaro, Gendreau, Tremblay & Oligny, 1998). Nouvion and colleagues found that proactive aggressive subjects had a greater incidence of personality disorders, including CD and APD, compared to reactive aggressive and non-aggressive control subjects (Nouvion, Cherek, Lane, Tcheremissine & Lieving, 2007).

These findings are in line with previous studies that have found proactive, but not reactive, aggression to be predictive of ODD, CD and externalising problems (e.g., Conner, Steingard, Anderson & Melloni, 2003; Pulkkinen, 1996; Vitaro et al., 1998). Given that individuals diagnosed with APD must have evidence of CD before the age of 15, early onset behavioural problems leading to adult antisocial behaviour was prevalent in the premeditated group. This provides evidence that premeditated-aggressive individuals may have increased personality psychopathology and be at increased risk for early aggressive and antisocial behaviours relative to impulsive-aggressive or non-aggressive individuals. The fact that CD was a distinguishable factor between groups is of significance due to the fact that CD is stable over time (Bassarath, 2001a; Kazdin, 2000), and is associated with criminal behaviour and substance abuse (Hser, Grella, Collins & Teruya, 2003; Mueser et al., 2006; Tcheremissine & Lieving, 2006).

Heilbrun et al. (1978) found that murderers whose violence was classified as impulsive were more likely to fail on parole that those whose murders were premeditated in nature. In a pharmacological treatment study comparing aggressive subtypes, inmates whose aggressive behaviour was classified as impulsive showed significant reductions in the frequency and intensity of aggressive acts, normalisation of event-related potentials (P3) and improvement in mood state measures during a six week trial of anticonvulsant phenytoin (Dilantin) when compared to placebo (Barratt et al., 1997a). Inmates whose aggressive behaviour was classified as premeditated in nature showed no improvement during the same trial. Similarly, Malone et al. (1998) examined the effect of lithium carbonate in CD children whose repeated aggressive behaviour was categorised as either predatory or affective in nature. Treatment response was associated with affective rather than predatory aggressive behaviour.

While distinguishing between these forms of aggression can not only lead to a better theoretical understanding of aggression (Coie & Dodge, 1998; Poulin & Boivin, 2000; Vitiello & Stoff, 1997), it can also to better prognostication. Such a distinction is also assumed to lead to the development of more specific interventions and treatment, which may then prove more effective than interventions aimed at aggression in general (McAdams, 2002; Vitello & Stoff, 1997).

Chapter 3

Prefrontal Cortex & Aggression

Research into the antecedents of violence and aggression indicates that there are many factors which contribute to the development of these behaviours. It is important to note that while there are general predictors of violent and aggressive behaviour, no single theory can account for causation in all situations. It is accepted that the causes of aggression are multi-faceted and that neurological deficit may be a factor in only a small percentage of those who demonstrate such behaviour. However, given that aggression – like any behaviour – ultimately derives from the normal and abnormal operations of the brain, closer examination of the aspects of brain structure and function relevant to aggressive behaviour are required.

Numerous studies, in both animals and humans, have supported an association between abnormalities in brain function and aggressive and violent behaviour (Filley et al., 2001; Golden, Jackson, Peterson-Rohne & Gontkovsky, 1996; Krakowski, 2003). Case studies of patients with neurological disorders or those who have suffered traumatic brain injury provide provocative insights into which brain regions, when damaged, might predispose to irresponsible, aggressive behaviour. Psychophysiological and neuropsychological assessments have also demonstrated that violent and/or aggressive individuals have lower brain functioning than controls, including lower verbal ability and diminished executive functioning (Barratt et al., 1997b; Dolan & Park, 2002; Hoaken et al., 2007).

The availability of new functional and structural neuroimaging techniques, such as PET, magnetic resonance imaging (MRI) and functional MRI (fMRI), has broadened our understanding of the neural circuitry that underlies aggressive and

antisocial behaviours. More specifically, as reviewed by Davidson and colleagues, a circuit that includes several regions of the prefrontal cortex, the amygdala, hippocampus, hypothalamus, anterior cingulate cortex, ventral striatum, and other interconnected structures has been implicated in various aspects of emotion regulation and affective style (Davidson & Irwin, 1999; Davidson, Jackson & Kalin, 2000a). Emotion regulation includes those processes which amplify, attenuate, or maintain an emotion, and thus incorporates the expression of aggressive behaviours. Related to this is evidence which suggests that individuals who are vulnerable to faulty regulation of negative emotion may be at increased risk for aggression and/or violent behaviour (Davidson, Putnam & Larson, 2000b).

Prefrontal cortical dysfunction has been implicated as a possible anatomical correlate of aggressive behaviour (Convitt et al., 1996; Critchley et al., 2000; Pietrini, Guazzelli, Basso, Jaffe & Grafman, 2000; Raine et al., 1994). Both cognitive and behavioural similarities have been noted between individuals who have frontal lobe damage and those who show characteristics of antisocial behaviour (Price, Daffner, Stowe & Mesulam, 1990). For example, Eslinger and Damasio (1985) noted that damage to the prefrontal area is associated with heightened aggression, emotional outbursts, disorganisation, and risk-taking behaviour. This observation has been a major impetus for the research on neuropsychological abnormalities in antisocial individuals. In particular, damage to the prefrontal regions of the brain and the resulting impairment in executive functions is considered to be associated with increased aggressive and antisocial behaviour (Brower & Price, 2001; Tateno, Jorge & Robinson, 2003).

3.1.1 The prefrontal cortex

The frontal cortex encompasses the brain areas anterior to the central sulcus and comprises approximately one third of the cerebral cortex. The frontal cortex can be divided into three principle regions: the primary motor cortex, the prefrontal cortex, and the limbic cortex (Duke & Kaszniak, 2000). The prefrontal cortex refers to the most anterior regions of the frontal lobes and it is functionally and anatomically heterogeneous (Fuster, 2001). The prefrontal cortex has a rich supply of connections with other neural regions. Cortically, it is connected with association cortex in the temporal, parietal and occipital lobes, and subcortically with the hippocampus, amygdala, thalamus, hypothalamus, subthalamus, septum, striatum, pons, and mesencephalon (Fuster, 2001; Pandya & Barnes, 1987). Given that the prefrontal cortex is connected to more brain regions that any other cortical region, its position allows the integration of information processed at lower levels, including input from the limbic circuits, as well as being the major target of the basal ganglia-thalamocortical circuits (Royall et al., 2002).

The prefrontal cortex, along with its underlying subcortical regions, is extensively interconnected with the major sensory and motor systems of the brain. Connections from the posterior cortical areas, particularly areas of multimodal convergence, bring information regarding the external environment. Subcortical pathways, including the amygdala, hippocampus, midbrain area, and thalamus, bring details about internal states (Duke & Kaszniak, 2000).

The prefrontal cortex has direct neural projections from a variety of limbic structures that are directly linked to the amygdala. There are input projections from the thalamus to the prefrontal cortex, and these connections contain information arising from the temporal cortex and the amygdala. Direct reciprocal connections

from the prefrontal cortex to the amygdala have also been identified (Afifi & Bergman, 1998). Output projections to the amygdala are both excitory and inhibitory in nature. However, damage to the prefrontal cortex results in an overactivation of the amygdala, suggesting that the effect of the prefrontal cortex on the amygdala is predominantly inhibitory (Gerwitz, Falls & Davis, 1997; Morgan, Romanski & LeDoux, 1993). With regard to the functionality of this connection, lesions to the prefrontal cortex in rats reduce the prefrontal inhibitory action on the amygdala, resulting in an increased difficulty in the extinction of aversive responses (Morgan et al., 1993), as well as impairing the ability to anticipate future negative consequences (Bechara, Tranel, Damasio & Damasio, 1996). Therefore, it appears that the prefrontal cortex plays an important role in regulating the acquisition of new responses, and the extinction of aversive responses (Lopez, Vazquez & Olson, 2004).

The prefrontal cortex also plays a central role in many aspects of social cognition (Rilling et al., 2002), including perspective taking (Frith & Frith, 1999), and also in the regulation of emotions such as aggression (see Blair, 2004 for review). Early descriptions of frontal lobe syndromes arose from several 19th century investigators, described in a number of reviews (Damasio et al., 1994; Macmillan, 2002; Tranel, Anderson & Benton, 1994), highlighting the changes displayed in social behaviour, personality, and emotional regulation that occurred after frontal lobe pathology. Subsequent investigators continued to elaborate on the nature and extent of these deficits, their causes and management (Miller & Cummings, 1999; Stuss & Knight, 2002), firmly establishing a vital role for the frontal lobes, particularly the prefrontal cortex, in such processes.

In an overview of neuroanatomy and neuropathology, Stuss and Benson (1984) described six specific manifestations of prefrontal damage: (1) inability to use

knowledge to regulate behaviour; (2) impaired ability to handle sequential behaviour; (3) impaired ability to establish or change a mental set; (4) impaired ability to maintain a mental set; (5) impaired ability to monitor personal behaviour; and (6) attitudes of apathy.

3.2 Prefrontal divisions

For clinical purposes, the prefrontal cortex can be divided into three distinct neuroanatomical regions: 1) dorsolateral prefrontal cortex (Brodmann's areas 9, 10, 46); 2) medial prefrontal cortex (including the functionally related anterior cingulate cortex and Brodmann's area 24); and 3) orbital prefrontal cortex (Brodmann's areas 11 and 12), corresponding to the most inferior and ventral parts of the prefrontal cortex (behind the eyes, or orbits). Both medial prefrontal and orbitofrontal are part of a frontostriatal circuit that has strong connections to the amygdala and other parts of the limbic system. Consequently, these regions are anatomically well suited for the integration of affective and non-affective information, and for the regulation of appetitive/motivated responses. Functionally, these regions are often considered together, as when researchers focus on effects of damage to ventromedial prefrontal cortex (Happaney et al., 2004).

The prefrontal cortex is a heterogeneous region of the brain and the three principal frontal-subcortical circuits are involved in cognitive, emotional, and motivational processes. The primary focus of the current research will be on the roles of the dorsolateral and orbital divisions of the prefrontal cortex, which manifest quite distinct anatomical and functional properties (Fuster, 1989; Stuss & Benson, 1986).

The *dorsolateral prefrontal cortex* projects primarily to the dorsolateral head of the caudate nucleus, which receives input from the posterior parietal cortex and

premotor areas. The dorsolateral circuit then connects to the dorsolateral part of the globus pallidus and rostral substantia nigra reticulate, and continues to the parvocellular area of the medial dorsal and ventral anterior portions of the thalamus. Projections from the thalamus back to the dorsolateral prefrontal circuit close the circuit (Cummings, 1993).

Functionally, the high-level cognitive abilities mediated by the dorsolateral prefrontal cortex and its connections are those referred to as „executive functions', including cognitive flexibility, temporal ordering of events, planning, monitoring and inhibiting pre-programmed behaviour, set-shifting, working memory and concept formation (Smith & Jonides, 1999). According to Cummings (1995), dysfunction in the dorsolateral prefrontal circuit is associated with circuit-specific problems including decreased verbal fluency, perseveration, difficulty shifting set, poor recall/retrieval of information, reduced mental control, limited abstraction ability, and poor response inhibition. However, while patients with lesions restricted to this region are concrete and perseverative and show impairments in reasoning and mental flexibility (Benton, 1986), they typically demonstrate intact perception, calculation, language abilities and storage of memories (Duke & Kaszniak, 2000).

The *orbitofrontal cortex* occupies the ventral region of the prefrontal cortex (Kringelbach & Rolls, 2004), which is reciprocally connected with the amygdala (Ghashghaei & Barbas, 2002). The orbitofrontal cortex projects to the ventromedial caudate nucleus, which receives input from other cortical association areas and brainstem regions, and has open interconnections with the dorsolateral prefrontal cortex, the temporal pole, and the amygdala (Davis & Whalen, 2001). The orbitofrontal cortex contains the secondary taste cortex, in which the reward value of taste is represented. It also contains the secondary and tertiary olfactory cortical areas,

in which the identity and also the reward value of odours are represented. The orbitofrontal cortex also receives information about the sight of objects from the temporal lobe cortical visual areas (Rolls, 1999).

The orbitofrontal-subcortical circuit is said to underlie social behaviour and appears to play a critical role in the representation of the reward value of a stimulus and the way in which this representation guides goal-directed behaviour (Rolls, 1999). Lesions specific to the circuit have been found to result in marked changes in personality, including disinhibition, impulsivity, and antisocial behaviour, and irritability and lability are often prominent (Cummings, 1995). Some of the changes may be related to difficulty in the learning and reversal of stimulus-reinforcement associations, and thus the correction of behavioural responses when they are no longer appropriate due to changes in reinforcement contingencies (Rolls, 2004; Hornak et al., 2004). Indeed, investigations in macaques have shown that lesions to the orbitofrontal cortex impair reversal learning (Dias et al., 1996a). Consistent with this, the orbitofrontal cortex is activated by monetary rewards and punishments, and the magnitude of the reinforcers (O'Doherty, Kringelbach, Rolls, Hornak & Andrews, 2001). The visual input to neurons in the orbitofrontal cortex is in many cases the reinforcement association of visual stimuli, one of which is information about faces. Such facial stimuli convey information that is important in social reinforcement (Rolls, 2004).

The *medial circuit*, begins in the anterior cingulate and projects to the nucleus accumbens. The anterior cingulate has interconnections with dorsolateral prefrontal cortex and the amygdala, and it also receives input from the ventral tegmental area (Duke & Kaszniak, 2000). The medial frontal-subcortical circuit is involved in

motivation. Lesions to this region often produce apathy, lack of motivation, decreased social interaction, and psychomotor retardation (Sbordone, 2000).

The *ventromedial* prefrontal region includes the medial and varying sectors of the lateral orbitofrontal cortex, encompassing Brodmann's areas 25, lower 24, 32, and medial aspect of 11, 12, and 10, and the white matter subjacent to all of these areas (Bechara, 2004). Patients with bilateral lesions of the ventromedial cortex develop severe impairments in personal and social decision-making, in spite of otherwise largely preserved intellectual abilities. Following damage to this region of the prefrontal cortex, patients develop difficulties in daily and future planning, and difficulties in choosing friends and activities (Bechara, Damasio & Damasio, 2000a; Bechara, Tranel & Damasio, 2002).

The identification of these adjacent circuits provides insight as to the similarities of behavioural changes caused by lesions to different brain regions. Whilst focal lesions to the areas of the prefrontal cortex have led to what have been labelled "frontal lobe syndrome", the involvement of multiple circuits in subcortical lesions has resulted in variable behavioural manifestations (Cummings, 1995). For example, studies of lesions to the globus pallidus have described patients with marked changes in personality and reduced activity levels with memory and executive function deficits, but with normal intelligence and language abilities (e.g., Strub, 1989).

In summary, the frontal–subcortical circuits are extensively connected to each other at the level of the frontal lobes. The circuits are discrete in subcortical regions. The dorsolateral circuit, because of its neuroanatomy, is uniquely able to integrate information from all three frontal–subcortical circuits. Here, the integrated information from the external world and the cognitive and emotional states of the individual can be used in the production of social behaviour.

3.3 Prefrontal dysfunction and aggression

Frontal lobe dysfunction in particular, has been invoked to explain the actions of individuals convicted of violent crimes, who appear to fail to inhibit impulsive, trivially motivated, or habitual aggression. Case studies as far back as 1935 have reported the onset of antisocial personality traits after frontal lobe injury (Blumer & Benson, 1975). Such cases typically involve damage to the orbitofrontal cortex, which clinical observations have associated with poor impulse control, explosive aggressive outbursts, inappropriate verbal lewdness, jocularity, and lack of interpersonal sensitivity (Duffy & Campbell, 1994). This dysregulation of affect and behaviour may occur while cognitive, motor, and sensory functioning remains relatively intact (Mesulam, 1986).

3.3.1 Lesion studies

Research on individuals who have suffered traumatic brain injury is of key importance in investigating the neural substrates of aggressive behaviour. The critical role of the prefrontal cortex in aggressive behaviour was initially recognised by case reports that prefrontal brain lesions could result in the emergence of antisocial behaviours or psychopathic traits in previously normal subjects (Damasio, Tranel & Damasio, 1990). A prime example of this disinhibition is found in the often cited case of Phineas Gage, a dependable and responsible stable railroad worker who was injured by a tamping rod that penetrated his skull through his orbital frontal cortex. After the accident he became irresponsible and impulsive, despite preserved general cognitive and motor skills (Damasio et al., 1994).

McAllister and Price (1987) evaluated 20 psychiatric patients with diverse types of frontal cortical pathology as identified by computed tomography scans and

EEG. Results indicated that 60% of the patients displayed disinhibited behaviour with affective lability and 10% displayed violent outbursts. However, the results of this study are difficult to interpret given that, in addition to having a frontal lobe pathology, all of the patients had at least one psychiatric diagnosis, and the exact neuroanatomical location of the pathology for each patient was not reported. In another study, Heinrichs (1989) found that a frontal cortical lesion was the best predictor of violent behaviour in a sample of 45 neuropsychiatric patients. Again, many of the patients in this study had other psychiatric diagnoses and the exact anatomical locations of the frontal neuropathologies were not specified.

Further data from neurological case reports have provided much useful information regarding the relationship between prefrontal cortical functioning and aggression. Thompson (1970) reported a case of a 33-year-old male with a history of violent behaviour subsequent to a head injury at the age of 12. A pneumencephalography revealed bilateral cortical atrophy in the prefrontal regions. Price et al. (1990) studied the adult behaviour patterns of two patients who acquired brain damage during childhood. While both patients developed relatively normally until the damage was sustained, following the damage these patients displayed an inability to respond to punishment or delay gratification, irresponsibility, sexual promiscuity, grand larceny, drug involvement, angry outbursts, arson, suspected rape, and physical violence. Although the exact location of the lesions in both cases is equivocal, neurological and neuropsychological examination indicated bilateral lesions in the prefrontal cortex.

Boone et al. (1988) described a 13-year-old female suffering from partial complex seizures localised primarily in the prefrontal cortex. Six weeks before being admitted to hospital she began exhibiting prominent behavioural changes consisting

of bizarre speech, sexual disinhibition, disobeying parental orders, and verbal and physical aggression. An EEG revealed activity in the frontal lobes and neuropsychological tests demonstrated deficits on prefrontal tests involving attention, alternation between tasks, performance on mazes, response inhibition, and distractibility. In a similar case, Eslinger and Damasio (1985) noted personality changes in patient EVR subsequent to surgical ablation of the orbital and mesial areas of the prefrontal cortex. Following the surgery, while his level of intelligence was above average, EVR began to engage in what the authors termed ‚sociopathic behaviour', including difficulties in decision-making, adjustment problems, poor judgement, and employment problems. The patient performed well on prefrontal cortical tests such as the WCST which the authors attributed to the fact that the dorsolateral prefrontal regions and superior mesial regions were left undamaged.

Meyers, Berman, Scheibe and Hayman (1992) noted similar behavioural sequelae involving disinhibition, poor judgement, and irresponsibility subsequent to surgical damage to the left orbital prefrontal cortex in a 33-year-old male. Interestingly, this patient performed in the above average range on prefrontal cortical tests such as the WCST; however this is again likely due to the preservation of the dorsolateral prefrontal cortex. These findings reflect those of Phineas Gage described earlier, who subsequent to his injury, was described as being untrustworthy, irresponsibly, and disrespectful. Again, the majority of the neural damage was located in the orbital and mesial prefrontal regions, whereas the dorsolateral area was found to be spared (Damasio et al., 1994).

Of the eight studies reviewed in the preceding section, six document a relationship between prefrontal cortical pathology and aggressive behaviour. However, none of these reports specified the exact location of the neuropathology

within the frontal lobes. As a result, these data do not provide evidence to implicate more specifically the dorsolateral or orbital prefrontal regions in the expression of aggressive behaviour. The remaining two reports indicate that their patients had lesions in the orbital prefrontal cortex and not the dorsolateral area.

Other case studies of patients who have sustained damage to the orbitofrontal region, such as EVR, resemble Gage in manifesting a behavioural profile that has been referred to as „acquired sociopathy' (Saver & Damasio, 1991; Tranel, 1994; Meyers et al., 1992; Blair & Cipolotti, 2000). Damasio et al. (1994) describe acquired sociopathy as a reactive, emotionally driven violence toward a person that is related to emotional inhibitory dyscontrol. Although showing minimal impairments on standard neuropsychological tests of intelligence and executive functions, these patients display marked deficits in real life tasks involving judgement, awareness of socially appropriate conduct, and the capacity to assess future consequences (Bechara et al., 2000b).

Blair and Cipolotti (2000) reported on JS who sustained damage to the orbitofrontal cortex and some damage to the left amygdala. Premorbidly, JS was described as being a quiet, withdrawn person who was never aggressive. Following the damage, JS showed unpredictable, impulsive-aggression and violence, and demonstrated deficits in the recognition of facial expression, particularly in the recognition of anger and disgust. He also produced significantly lower skin conductance responses (SCR) to the anger and disgust expressions compared with comparison groups.

Further clinical data demonstrate that lesions in the orbitofrontal cortex and adjacent prefrontal regions produce syndromes characterised by impulsivity and aggression. Anderson et al. (1999) reported on two individuals, tested in their

twenties, who suffered early damage to orbital and lateral sectors of the prefrontal cortex. Both exhibited a significant deficit in moral reasoning, a history of verbal and physical aggression, and intermittent, explosive bursts of anger. A further study of two adults who sustained frontal lobe injury in childhood suggests that early damage to orbitofrontal regions may lead to a "comportmental learning disability" that closely resembles sociopathy and includes a diminished capacity to inhibit violence (Price et al., 1990).

Further evidence implicating the orbitofrontal regions comes from a large retrospective study of Vietnam veterans with penetrating head injuries, which found that ventromedial frontal and orbitofrontal lesions, as assessed by computed tomography scans, specifically increased the risk of aggressive and violent behaviour. (Grafman et al.,1996). Data have also been reported showing higher rates of antisocial behaviour (including stealing, physical assault and sexual comments or advances) in patients with frontotemporal dementia, even when compared with equally cognitively impaired patients with Alzheimer's disease (Miller, Darby, Benson, Cummings, & Miller, 1997; Stip, 1995).

3.3.2 Neuroimaging and clinical neurological findings

Brain imaging studies are now beginning to confirm the role of the prefrontal cortex in modulating and controlling violence in humans. Reviews of brain imaging studies of violent and psychopathic populations completed by Raine (1993), Mills and Raine (1994), Raine and Buchsbaum (1996), Henry and Moffitt (1997), and Bufkin and Luttrell (2005), while showing some variability across studies, concur in indicating that violent offenders have functional deficits in the anterior regions of the brain, particularly the frontal region.

Most recently, in their review of 17 neuroimaging studies, Bufkin and Luttrell (2005) found that the areas associated with aggressive and/or violent behaviour, particularly impulsive acts, are located in the prefrontal cortex and the medial temporal regions. Of the 17 studies reviewed, 14 specifically examined possible links between frontal lobe pathology and aggressive and/or violent behaviour. In the 10 single photon emission computed tomography (SPECT) and PET studies, 100% reported deficits in either prefrontal (8 of 10 studies) or frontal (2 of 10 studies) functioning in aggressive, violent and/or antisocial groups compared to non-aggressive patients or healthy controls. Analyses of specific regions in the prefrontal cortex revealed that individuals who were aggressive and/or violent had significantly lower prefrontal activity in the orbitofrontal cortex (4 of 10 studies), anterior medial cortex (2 of 10 studies) and/or superior frontal cortex (1 of 10 studies). In the four MRI studies, half reported decreased grey matter volume in prefrontal or frontal regions, and 25% reported non-specific white matter abnormalities, not localised to the frontal cortex.

Initial studies demonstrate anterior brain dysfunction in individuals with a history of violence. Goyer et al. (1994), using PET in an auditory activation condition, showed that an increased number of aggressive impulsive acts were associated with reduced glucose in the anterior medial and left anterior orbitofrontal frontal cortex of 17 personality disordered patients. Volkow and colleagues in two PET studies which compared forensic psychiatric patients with normal controls, documented decreased frontal cortical blood flow or metabolism associated with „repetitive' and „purposeless' violent behaviour (Volkow & Tancredi, 1987; Volkow et al., 1995). Søderstrom et al. (2000) using SPECT, found reduced blood flow in both frontal and temporal lobes of 21 individuals convicted of impulsive violent crimes.

In a PET study evaluating responses to the probe metachlorophenylpiperazine, decrements in the lateral, medial and orbital frontal cortices were found at baseline in men with a history of physical aggression and in the orbital frontal cortex for both men and women with a history of physical aggression (New et al., 2009). Furthermore, a series of studies demonstrated that while normal subjects show increased relative glucose metabolism in orbitofrontal cortex and anterior cingulate gyrus following acute serotonergic stimulation, impulsive-aggressive personality disordered patients show decreased relative metabolism in this area (New et al., 2002; Siever et al., 1999; Soloff et al., 2003). These studies suggest that orbitofrontal and adjacent regions may exert an inhibitory influence on aggression, perhaps through a serotonergic mechanism.

Interictal episodes of impulsive aggression have also been observed in patients with temporal lobe epilepsy. Such patients who display episodes of impulsive aggression have a highly significant reduction (approximately 17%) in left prefrontal gray matter compared with temporal lobe epilepsy patients with no history of aggression or controls (Woermann et al., 2000).

One particular set of studies was undertaken by Raine and colleagues on a heterogeneous group of suspected murderers pleading not guilty by reason of insanity. In a first study Raine et al. (1994) conducted a PET study on 22 subjects accused of murder and 22 matched controls. The offender group had significantly lower glucose metabolism in both medial and lateral prefrontal cortex relative to the controls. Raine, Buchsbaum and LaCasse, (1997) in a study of 41 murderers found hypoactivation in prefrontal territories including lateral and medial regions of the prefrontal cortex, as well as activation in the right amygdala, compared with age- and sex-matched controls. In a subsequent reanalysis of these data, murderers were classified as those

who commit planned, predatory murder or those who committed affective, impulsive murder. The impulsive murderers showed reductions in lateral prefrontal metabolism compared with controls, whereas the predatory group did not (Raine et al., 1998). Findings from a more recent structural MRI study indicated that individuals with a diagnosis of APD recruited from the community showed an 11% reduction in the volume of gray matter in the prefrontal cortex, compared to both normal controls and a substance dependence control group (Raine et al., 2000).

A number of studies have also found abnormal frontal EEG activity, as well as diminished frontal event related potentials (ERP), correlating with antisocial personality disorder or histories of aggression (Bauer, O'Connor & Hesselbrock, 1994; Bernat, Hall, Steffen, & Patrick, 2007; Finn, Ramsey & Earleywine, 2000; O'Connor, Bauer, Tasman & Hesselbrock, 1994).

3.4 The specific roles of the orbitofrontal and dorsolateral prefrontal cortex in aggression

The cumulative evidence from the studies reviewed above indicates that clinically significant frontal lobe dysfunction is associated with aggressive behaviour. Subjects with both traumatic brain injury and neurodegenerative disorders primarily involving the prefrontal cortex display increased rates of aggressive and antisocial behaviour compared to subjects who have no, or non-frontal brain injury. Studies employing neuropsychological testing, neurological examination, EEG, and neuroimaging have also found evidence for increased rates of prefrontal network dysfunction among aggressive and antisocial subjects (see Brower & Price, 2001; Bufkin & Luttrell, 2005 for reviews).

Unfortunately, however, the above studies have placed little emphasis on considering the separable regions of frontal cortex. Data based on acquired damage to the prefrontal cortex implicate the ventromedial and orbitofrontal regions of the prefrontal cortex (Damasio et al., 1994; Grafman et al., 1996). Alternatively, impairments in the dorsolateral region, which is critically involved in cognitive flexibility and response perseveration, cannot be ruled out because repetitive aggressive behaviour can be conceptualised as perseverative, unmodifiable behaviour in response to repeatable punishment. One of few studies to dissociate functional regions of the prefrontal cortex with regard to aggression was conducted by Goyer et al. (1994) who found lower normalised cerebral blood flow (CBF) in lateral orbitofrontal cortex that correlated with a history of reactive aggression.

The parallels between the effects of orbitofrontal lesions on social behaviour and the symptoms of antisocial disorders are prominent. In addition to the classic case of Phineas Gage, further studies of individuals with orbitofrontal cortex lesions have described these patients as disinhibited, socially inappropriate, misinterpreting others' moods, impulsive, unconcerned with the consequences of their actions, irresponsible in everyday life, lack insight, and show a poor sense of initiative (Rolls et al., 1994). Based on these findings, a logical prediction is that the orbitofrontal cortex activity in response to provocation may be attenuated in certain individuals, predisposing to aggression and violence (Davidson et al., 2000b).

Several hypotheses about the role of the orbitofrontal cortex in behaviour regulation have been developed. One of these, the „somatic markers hypothesis' suggests that ventromedial frontal lobe lesions impair the capacity to consider emotions when making a decision (Damasio, 1996). Alternate and often complementary hypotheses suggest that the primary deficit following such frontal

lobe lesions lies in self-reflective awareness (Stuss, Gow & Hetherington, 1992), perspective taking (Stuss, Gallup & Alexander, 2001b), social schema knowledge (Grafman et al., 1996), the ability to respond appropriately to social reinforcers (Rolls et al., 1994), and the ability to make inferences about the mental state of others („Theory of Mind'; Stone, Baron-Cohen & Knight, 1998).

3.5 The role of the prefrontal cortex in impulsive- and premeditated-aggression

Despite increasing support for the impulsive-premeditated aggression distinction as outlined in the previous chapter, surprisingly little is known about the neurophysiological and neuroanatomical factors that characterise these subtypes of aggression in humans. Indications can nevertheless be gained from the literature on the cortical and subcortical mechanisms thought to be involved in aggression and violence per se.

At a cortical level, it has been proposed that abnormalities of the anterior cingulate cortex and orbitofrontal cortex, regions of the ventromedial prefrontal cortex, alone or in combination with abnormalities of the amygdala, underlie the hyperarousal and dyscontrol states seen in impulsive aggressors (Best, Williams & Coccaro, 2002; Blair, 2004; Davidson et al., 2000b; New et al., 2002, 2004). Blair and Davidson et al. proposed that the anterior cingulate cortex and orbitofrontal cortex are normally activated during anger arousal via serotonergic mechanisms and exert inhibitory influence over aggressive emotional responding via mechanisms including inhibition of the amygdala, hypothalamus, and brainstem periaqueductal gray.

Certainly, damage to medial frontal and orbitofrontal cortex is associated with increased risk for the display of impulsive-aggression in humans whether the lesion occurs in childhood (Anderson et al., 1999; Pennington & Bennetto, 1993), or

adulthood (Grafman et al., 1996). In addition, neuroimaging data have revealed reduced frontal functioning in patients presenting with reactive aggression (Søderstrom et al., 2000; Volkow & Tancredi, 1987; Volkow et al., 1995). Further studies of impulsive-aggression have found hypoactivation of the anterior cingulate cortex and orbitofrontal regions of the prefrontal cortex (Best et al., 2002; New et al., 2002). Interestingly, this reduced frontal functioning is not observed in patients presenting with predominantly premeditated-aggression (Raine et al., 1998). This is consistent with neuropsychological data that indicate that psychopathic individuals, individuals who present with marked instrumental aggression, do not present with poor performance on general measures of frontal lobe functioning (LaPierre, Braun, & Hodgins, 1995; Mitchell et al., 2002).

It has been suggested that regions of the orbitofrontal cortex are involved in a system that is crucial for social cognition and the modulation of impulsive-aggression (Blair, 2004; Blair & Cipolotti, 2000). The orbitofrontal cortex receives highly processed sensory information concerning an individual's environmental experience (Mesulam, 1986) and is hypothesised to play a role in the perception of social signals, in particular, facial expressions of anger (Blair et al., 1999). Rolls (2000) suggested that the orbitofrontal cortex modulates the subcortical systems mediating impulsive-aggression through the expectations of reward and identifying if these expectations have been violated. He argued that frustration, which has been linked to the display of impulsive-aggression, occurs following the initiation of a specific behaviour to achieve an expected reward and the subsequent absence of this reward. It can therefore be suggested that orbitofrontal cortex may increase neuronal activity in the subcortical systems mediating impulsive-aggression when an expected reward has not been achieved and suppress neuronal activity when the expected reward is achieved.

Blair and Cipolotti (2000) proposed a further process termed Social Response Reversal (SRR). The position stresses the role of social cues in modulating social behaviour (Blair, 2001; Blair & Cipolotti, 2000). The SRR is thought to be activated by another individual's angry expression, other negative valenced expressions and situations associated with social disapproval. In line with this, the orbitofrontal cortex is activated by negative emotional expressions including anger, fear and disgust (Blair et al., 1999; Kesler-West et al., 2001; Sprengelmeyer, Rausch, Eysel & Przuntek, 1998). Moreover, patients with orbitofrontal lesions are impaired in their ability to recognise facial expressions, particularly anger (Blair & Cipolotti, 2000; Hornak et al., 1996).

In a discussion of the literature, Davidson et al. (2000b) suggested that individuals can typically regulate their negative affect and can also profit from restraint-producing cues in their environment, such as others' facial expression of fear or anger. Information about behaviours that indiciate threat (e.g., hostile stares, threatening words) is conveyed to the amygdala, which then projects to other limbic structures, and it is there that information about social context derived from the orbitofrontal projections is integrated with one's current perceptions. The orbitofrontal cortex, through its connections with other prefrontal sectors and with the amygdala, thus plays an important role in inhibiting impulsive-aggressive outbursts because prefrontal activations that occur during anger arousal constrain the impulsive expression of emotional behaviour.

Davidson et al. (2000b) further proposed that dysfunctions in one or more of these regions and/or in the interconnections among them may be associated with faulty regulation of negative emotion and an increased propensity for impulsive-aggression. Firstly, people with prefrontal and/or amygdala dysfunction might

misinterpret environmental cues, such as facial expressions of others, and react impulsively to a misperceived threat. According to Albert, Walsh and Jonik (1993), most acts of human aggression are a reaction toward a threat, be it real or imagined. Therefore, the perception of whether a stimulus is threatening is vital in the cognitive processing leading to aggressive behaviour. Secondly, evidence suggests that individuals vary considerably in their ability to suppress negative emotion. Therefore, individuals with decreased prefrontal activity may have greater difficulty suppressing negative emotions than those individuals who have greater prefrontal activation.

3.6 Subcortical Structures

Subcortically, four structures are viewed as important in mediating aggressive behaviour: the amygdala, hippocampus, midbrain area, and thalamus. The amygdala, hippocampus and prefrontal cortex make up part of the limbic system governing the expression of emotion, while the thalamus relays inputs from subcortical limbic structures to the prefrontal cortex (Mirsky & Siegel, 1994). Traditionally, subcortical and limbic regions of the brain have been viewed as involved in the generation of aggressive feelings and behaviours, while the prefrontal cortex is viewed as inhibiting and modulating these basic emotions (Weiger & Bear, 1988). Consequently, it could be argued that it is the relative balance of activity between the prefrontal and subcortical brain regions which may be critically important in predisposing one to aggressive behaviour. If prefrontal functioning is reduced relative to subcortical structures, the individual may be more prone to aggression in general and perhaps impulsive-aggression in particular. Raine et al.'s (1998) results support this argument, finding lower prefrontal activity in affective (impulsive) murderers and higher subcortical activity in both affective and predatory murderers relative to controls.

3.7 Conclusion

The consistency across studies suggests that prefrontal dysfunction may underlie a predisposition to aggressive behaviour. Evidence is strongest for an association between prefrontal dysfunction and an impulsive subtype of aggressive behaviour (e.g., Bassarath, 2001b; Brower & Price, 2001; Raine et al., 1998). However, given the lack of specificity in research in aggression regarding the subtypes of impulsive- and premeditated-aggression, the mediating role of the prefrontal cortex in premeditated-aggression cannot be ruled out.

Chapter 4

Rationale

The aim of the present study is to test hypotheses related to information processing differences pertaining to prefrontal functioning between impulsive-aggressive and premeditated-aggressive individuals. According to the clinical literature, damage to the prefrontal cortex results in poor impulse control and explosive, aggressive outbursts (Duffy & Campbell, 1994). However, although problems of disinhibition are implicated, there are many ways in which prefrontal dysfunction may heighten the risk for aggressive behaviour.

Perhaps most salient are defects in executive functions. Such functions involve the ability to plan and problem-solve, deficits in which may lead to careless or inappropriate behaviours, interpersonal inappropriateness, as well as rigidity or difficulty modifying behaviour. A lessened capacity to self-correct, learn, and think flexibly, will have a particularly salient effect in situations which lack clear rules and structure as an inability to generate a suitable response may exacerbate frustration and thus the tendency to reflexive emotional responding.

A reliance on reflexive responding suggests a certain degree of behavioural rigidity. The behavioural rigidity which is often associated with frontal lobe damage manifests in overly persistent or perseverative responding (Lezak, Howieson & Loring, 2004). Such perseveration can be understood as the continuation of a response after it is no longer appropriate. An inability to adjust inappropriate behaviours in social situations may lead to heightened interpersonal conflict. Related to this notion is the ability to correctly interpret the emotion of others, that is, deficits in such abilities will also result in inappropriate responding in social interactions. The

inability to inhibit inappropriate behavioural responses may also have a particularly salient influence in propelling one toward aggressive responding. Prefrontal deficits may result in an inability to control the behavioural expression of mood changes, and thus the usual capacity to inhibit inappropriate emotional responses may break down.

Studies of patients with neurological disorders have provided provocative insights into which structural brain mechanisms, when damaged, may predispose some persons to antisocial and aggressive behaviour. However, while such individuals indicate a link between brain damage and the onset of antisocial behaviour, it could be argued that these findings have little relevance to those individuals in the community who have consistent aggressive behaviour throughout their lives, yet have not suffered gross brain damage. It has been speculated that such individuals possess more subtle prefrontal dysfunction than the blunt damage in acquired sociopaths, but there have been few tests of this hypothesis. Specifically, it is not known whether aggressive individuals in the community have subtle structural deficits in the prefrontal cortex in the absence of discernable lesions.

With the exception of Stanford et al. (1997), most neuropsychological research on aggression have investigated possible deficits in either incarcerated prison inmates or psychiatric patients, ignoring the large number of individuals in the general population who commit aggressive acts yet have not come into contact with the criminal justice or mental health systems. Such sampling biases inhibit the usefulness of such results in the development of interventions and treatments for reducing specific forms of aggression.

The current review has also emphasised the importance of distinguishing between predominantly impulsive-aggressive behaviour and predominantly premeditated-aggressive behaviour. While distinguishing between aggressive

subtypes is clearly important for understanding the causal features of such behaviour, it has also been shown to be crucial in relation to proper intervention and treatment (Pulkkinen, 1996; Vitaro et al., 1998). Furthermore, the results of Barratt et al. (1997a) and Malone et al. (1998) clearly suggest that the extent to which biological variables influence impulsive- and premeditated-aggressive behaviour differs. The problem is, however, as Scarpa and Raine (2000) highlighted, "few studies of the biological bases of antisocial behaviour in humans have categorised aggression or violence according to these subtypes" (p. 321). Rather, most have used heterogeneous groups of aggressive individuals comparing them to non-aggressive controls. This has led to equivocal and sometimes misleading results throughout the literature. In attempting to overcome these problems, the current study attempts to delineate the neurobiology of aggression through an investigation of the neuropsychological differences between predominantly impulsive- and predominantly premeditated-aggressive individuals.

Studies of subjects with acquired frontal lobe injury support the association between increased aggression and focal orbitofrontal, or ventromedial frontal injury, or both. The neuropsychological literature, however, tends to find increased aggressive behaviour associated with deficits in executive function, which correlate with dorsolateral prefrontal dysfunction (Dolan & Anderson, 2002; Stanford et al., 1997). The dorsolateral and orbitofrontal prefrontal cortex are parts of an integrative functional system and they typically work together, even in a single situation. Thus, one hypothesis to account for discrepant data is that orbitofrontal and dorsolateral prefrontal dysfunction contribute to aggressive dyscontrol in different ways.

Dorsolateral dysfunction may become evident through its impact on executive functions. Such executive deficits, as investigated in Study 1, may increase the risk of

aggression through its direct effects on impulse control, planning, self-monitoring, and cognitive flexibility. Individuals who have dysfunction involving the orbitofrontal cortex comprise a different group. Retrospective data strongly support a link between the disinhibited type of frontal network syndrome and aggressive dyscontrol. Thus, dysfunction in the orbitofrontal cortex may lead to aggression through impaired emotion recognition, as examined in Study 2, as well as through inhibitory, response reversal, and decision-making deficits investigated in Study 3 (Brower & Price, 2001).

Chapter 5

Study 1: Executive Functioning

Executive functioning has been conceptualised as the capacity to use certain „higher-order' cognitive abilities to adaptively regulate one's goal-directed behaviour (Giancola, Roth & Parrott, 2006). While previous notions of executive functioning have described a homogenous set of processes, not differentiating among individual skills, it is now understood that executive functions are best understood as an umbrella term, encompassing a number of interrelated subskills, necessary for purposive action (Stuss & Benson, 1986). One can thus conceive of executive functions as a set of processes that are distinct from one another but that nonetheless work together in order to meet a particular common goal (Sylvester et al., 2003).

In a review of neuropsychological assessment procedures, Lezak (1995) stated that executive functions are those capacities which enable a person to successfully engage in independent, purposeful, and self-serving behaviours. Stuss (1992) has proposed an integrated model of executive function, including a set of associated skills that allow the individual to develop goals, actively hold them in memory, monitor performance, and control for interference to achieve these goals. Shallice (1990) and Walsh (1978) fine-tuned the concept further, suggesting that executive functions may not be employed during the activation of well-learned routine behaviours, but are enlisted in novel or unfamiliar circumstances in which no previously established routines for responding exist. In other words, executive functioning allows humans to respond to situations in a flexible manner, to create and adapt plans and to base their behaviour on internally held ideas rather than being governed exclusively by external stimuli.

Such cognitive abilities subsumed under the rubric of „executive functioning' include attentional control, strategic goal planning, abstract reasoning, cognitive flexibility, hypothesis generation, temporal response sequencing, as well as the ability to organise and adaptively use information contained in the working memory (Fuster, 1989; Stuss & Benson, 1984, 1986). Thus executive dysfunction may be reflected by poor planning and organisation, difficulties generating and implementing strategies for problem-solving, perseveration, reduced self-control, impulsivity, inability to use feedback to correct errors, and rigid or concrete thought processes (Lezak, 1995; Stuss & Benson, 1986).

In daily life, individuals must rely on such processes when automatic or previously learned behaviours can no longer achieve a goal, for example, when there is a need to override habitual responses, solve new problems or shift between different tasks. This capability is crucial in changing environments where there is a constant need to adapt behaviour by detecting and focusing on the goal-relevant information and selecting the most appropriate behaviour (Mansouri, Tanaka & Buckly, 2009). Executive functions thus allow the individual to select and schedule appropriate sequences of actions when effective new plans of action have been formulated (Kempton et al., 1999).

5.1 Neuroanatomy of executive functions

Executive functions were thought to be subserved primarily, if not solely, by the frontal lobes since at least the 19th century when Phineas Gage demonstrated dramatic changes in self-regulatory function after a dynamic tamping rod was propelled through his frontal lobe (Damasio et al., 1994). There is now convincing evidence that executive functions depend, in large part, upon the integrity of the

prefrontal cortex (Stuss, 2002), as well as its connections with the parietal and temporal lobe structures, the limbic structures such as the amygdala and the hippocampus, and the striatum (Kempton et al., 1999). Damage in this area can consequently result in impairment in planning and problem-solving, impulsive, disinhibited or disorganised behaviour, cognitive rigidity, as well as a reduced ability to self-regulate and consider the outcomes of behaviours (Blair, 2004).

According to Royall et al. (2002), the role of the prefrontal cortex in executive functioning is due to its unique structure and pattern of connectivity. However, while the prefrontal cortex is the primary brain region related to executive functioning, it should be noted that such abilities are not governed solely by the prefrontal cortex and that this region of the brain is also responsible for other ‚non-executive' functions (Duffy & Campbell, 1994). The prefrontal cortex is richly and reciprocally interconnected with almost every cortical and subcortical neural region. Consequently, damage to these other areas adversely affects executive functioning (Fuster, 2001). Specifically, structures such as the anterior cingulate (Devinsky, Morrell & Vogt, 1995), the striatum (Owen et al., 1992), the thalamus (Alexander, Crutcher & DeLong, 1990), and the cerebellum (Allen, Buxton, Wong & Courchesne, 1997) are all involved, at least in part, in governing executive abilities. Nevertheless, while executive functions are not the sole domain of the prefrontal cortex, this region of neural tissue is considered to be the primary cortical substrate that subsumes these related cognitive functions. Looking more specifically at the prefrontal cortex, while research suggests that the orbitofrontal region of the prefrontal cortex is related to emotional regulation and impulsivity, thereby increasing susceptibility to aggressive impulses, the dorsolateral prefrontal cortex has primarily been linked to executive functions (Giancola, 1995; Mattson & Levin, 1990; Smith & Jonides, 1999).

Neuroimaging studies have demonstrated that many executive processes evoke activity in a network of brain regions (Miller & Cohen, 2001) that include the dorsolateral prefrontal cortex and related subcortical regions including the basal ganglia and thalamus, and selected temporal and parietal regions (Berman et al., 1995; Casey et al., 2001; Kirino, Belger, Goldman-Rakic & McCarthy, 2000; McCarthy, Luby, Gore & Goldman-Rakic, 1997; Monchi, Petrides, Petre, Worsley & Dagher, 2001). Converging evidence from patient studies have shown that damage to this network results in impairments to behavioural flexibility and to anticipation of future consequences (Knight & Stuss, 2002; Lhermitte, 1986).

Measures of executive functioning are typically derived from tests that assess programming and planning of goal-oriented motor behaviour skills, modulation of behaviour in light of expected future consequences, anticipation of events in regulating behaviour, learning contingency rules and using feedback cues, inhibition, cognitive flexibility, abstract reasoning, problem-solving, sustained attention and concentration (Cauffman, Steinberg & Piquero, 2005). Alvarez and Emory (2006) completed a qualitative review of three popular executive function measures: the WCST, phonemic verbal fluency, and the Stroop colour-word task. They found that these tests are sensitive, but not specific, to frontal lobe damage. Overall, they found that individuals with frontal lobe lesions perform more poorly than healthy controls on these tests, although several studies indicate that patients perform within normal limits (e.g., Ahola, Vikki & Servo, 1996; Damasio, 1994; Eslinger & Damasio, 1985; Heck & Bryer, 1986; Shallice & Burgess, 1991). These results suggest that such tests should not be used as frontal lobe tests per se, but rather as tests of specific executive functions such as problem-solving and cognitive flexibility. Stuss and Alexander

(2000) support this claim, arguing that "there is no frontal homunculus, no unitary executive function (p. 291).

5.2 Executive functioning measures

5.2.1 Verbal Fluency Test

The Verbal Fluency Test is used to assess the ability to update working memory and flexibility of verbal thought processes. Alvarez and Emory (2006) in their review of the task reported that ten (out of ten) studies found that persons with frontal lobe lesions produce significantly fewer words than healthy controls, and eight (out of nine) studies indicate that person with frontal lobe lesions perform worse than persons with non-frontal lobe lesions. In support of these findings, Henry and Crawford (2004) conducted a meta-analysis of 31 studies and found that individuals with focal frontal lesions had larger deficits in verbal fluency as compared to healthy controls.

Studies which have examined whether the frontal lobes are activated during verbal fluency performance in healthy adult populations have found increased activation in the left dorsolateral prefrontal cortex (Frith et al., 1995; Frith, Friston, Liddle & Frackowiak 1991; Warkentin & Passant, 1997), anterior cingulate (Frith et al., 1991; 1995; Phelps, Hyder, Blamire & Shulman, 1997), and left inferior frontal gyrus (Paulesu et al., 1997; Phelps et al., 1997). The findings of increased activation in frontal areas along with the finding of Parks et al. (1988) of increased overall frontal lobe activation in frontal areas suggest that an intact frontal cortex, especially in the left hemisphere, is required for verbal fluency performance. Further research has implicated the dorsolateral prefrontal cortex, more specifically, in verbal fluency functioning. rCBF and PET studies revealed significant flow augmentation and

increased activity in dorsolateral prefrontal cortex during word fluency tasks (Cantor-Graae, Warkentin, Franzen & Risberg, 1993; Warkentin, Risberg, Nilsson, Karlson & Graae, 1991).

5.2.2 Trail Making Test

The letter-number switching condition of the Trail Making Test is a neuropsychological measure of the ability to initiate, switch and stop a sequence of complex purposive behaviour that requires attention and concentration skills (Moffitt & Henry, 1989). This ability to think flexibly (also referred to as set shifting) is considered to be an integral component of executive functioning and has been found to be sensitive to frontal lobe damage (e.g., Boll, 1981). Zakzanis, Mraz, and Graham (2005) revealed distinct left-sided dorsolateral and medial frontal activity when comparing performance on Part B versus Part A of the trail making test in healthy young adults. Moll, de Oliveira-Souza, Moll, Bramati and Andreiuolo (2002) also found activation in the dorsolateral prefrontal cortex and supplementary motor area/cingulate sulcus in normal adults.

Stuss and colleagues, in earlier research, found that patients with damage in dorsolateral frontal areas were most impaired on Part B of the trail making test in comparison to patients with damage to other areas of the brain, including the medial and orbital regions of the frontal cortex (Stuss, Floden, Alexander, Levine & Katz, 2001a). These findings are in line with clinico-anatomic and functional neuroimaging data that point to a critical role of the dorsolateral and medial prefrontal cortices in the regulation of cognitive flexibility (e.g., Moll et al., 2002; Shibuya-Taoshi, et al., 2007; Szatkowska, Szymanska, Bojarski & Grabowska, 2007; Zakzanis et al., 2005).

5.2.3 Tower of Hanoi

Tower of Hanoi investigates planning and spatial problem-solving ability and is considered to be especially sensitive to frontal system dysfunction (Grafman et al., 1992; Goel & Grafman, 1995; Morris, Miotto, Feigenbaum, Bullock & Polkey, 1997). Using a simplified version of this task which he termed the Tower of London, Shallice (1982) demonstrated that certain patients with frontal lesions showed pronounced impairments in planning that could not be accounted for in terms of any more basic perceptual or memory problems. Further studies, some of which have used a computerised version of the Tower of London or Tower of Hanoi, have both replicated findings of deficits in patients with frontal lesions (Carlin et al., 2000; Goel & Grafman, 1995; Owen et al., 1990; Yochim, Baldo, Kane & Delis, 2009), and also demonstrated frontal activation on PET imaging when normal volunteers perform this task (Morris, Ahmed, Syed & Toone, 1993; Rowe, Owen, Johnsrude & Passingham, 2001; van den Heuvel et al., 2003).

More specific analyses of the separable regions of the prefrontal cortex involved in the Tower of Hanoi are limited, however research utilising the Tower of London indicate predominant dorsolateral involvement in this task. Owen, Doyon, Petrides and Evans (1996) found activation of left dorsolateral prefrontal cortex, as well as several activations in right premotor and parietal cortices that may be associated with visuo-spatial maintenance. Similarly, Baker et al. (1996) using PET found that performance was associated with bilateral premotor and dorsolateral frontal cortex, anterior cingulated gyrus, bilateral medial and superior parietal cortex, and lateral occipital cortex. They also found that increasing task difficulty was associated with relative increases in frontal, premotor, and medial parietal cortices and robust activations in right dorsolateral prefrontal and bilateral premotor cortex. Similarly,

Dagher, Owen, Boecker and Brooks (1999) found that increased task complexity correlated with increased activation in bilateral premotor, dorsolateral prefrontal, and rostral anterior cingulate cortex, and right dorsal caudate nucleus.

More recently, Lazeron et al. (2000) found increased activity in bilateral dorsolateral frontal cortex, left anterior cingulate cortex, left insula, and bilateral cuneus and precuneus and left angular gyrus. Similar results were also reported by Cazalis et al. (2003). Van den Heuvel et al. (2005) found that planning and task complexity on the Tower of London task were associated with activation in right and dorsolateral frontal cortex respectively.

5.2.4 Stroop Colour-Word Interference Task

A major role of the frontal lobes is to control response options (Fuster, 1997; Stuss, Shallice, Alexander & Picton, 1995) through marshalling inhibitory processes, establishing response selection, or maintaining constant activation of the intended goal. In general, brain imaging data support the sensitivity and specificity of the Stroop task to frontal lobe functioning. The Stroop task has been reported to distinguish patients with frontal lobe damage from healthy controls (Stuss et al., 2001a; Vendrell et al., 1995), and patients with lesions in other areas (Perret, 1974; Stuss et al., 2001a). Frontal patients also often fail to inhibit the dominant response in the interference condition of the task (Holst & Vilkki, 1998; Spreen & Strauss, 1998).

More specifically, the Stroop task has been consistently shown to recruit the anterior cingulate cortex as well as other frontal regions, particularly regions of the dorsolateral prefrontal cortex (see Botvinick, Cohen, & Carter, 2004; MacLeod & McDonald, 2000 for reviews). The anterior cingulate is a critical brain region for adequate performance on the Stroop task given its role in selective attention (Peterson

et al., 1999), and studies have shown that patients with lesions of the anterior cingulate cortex present with profound increases in the level of interference that they show during incongruent Stroop trials (Stuss et al., 2001a; Swick & Jovanovic, 2002).

Several studies also indicate that the Stroop test activates the middle frontal gyrus (Banich et al., 2000; Bush et al., 1998; Leung, Skudlarski, Gatenby, Peterson, & Gore, 2000; Peterson et al., 1999), medial frontal cortex, and dorsolateral prefrontal cortex (Evan Nee, Wager, & Jonides, 2007). Performance on the Stroop Test has also been found to be accompanied by right frontal activation as measured by PET (Bench et al., 1993; Taylor, Kornblum, Lauber, Minoshima, & Koeppe, 1997).

Evan Nee et al. (2007) completed a quantitative meta-analysis on 47 neuroimaging studies involving tasks purported to require the resolution of interference, including the Stroop, flanker, go/no-go, stimulus-response compatibility, Simon, and stop-signal tasks. They found activation in the right dorsolateral prefrontal cortex and left premotor/supplementary motor area which is consistent with the idea that these regions are implicated in the resolution of interference during response selection (Bunge, Hazeltine, Scanlon, Rosen & Gabrieli, 2002; Durston, Thomas, Worden, Yang & Casey, 2002a; Durston et al., 2002b; Praamstra, Kleine & Schnitzler, 1999). Activation was also found in the anterior cingulate cortex, which again highlights its function as a monitor involved in the resolution of response conflict (Botvinick, Braver, Barch, Carter & Cohen, 2001).

5.2.5 The Brixton Test

The Brixton Test is a concept attainment task. The task assesses mental flexibility, the ability to use feedback to shift cognitive sets and goal-directed behaviour. This class of test, of which the WCST is the most well-known example, is

known to present problems for patients with frontal lobe lesions (Stuss et al., 2000). By comparison with the WCST, the Brixton Test improves the potential discriminability of different patterns of functional deficit (e.g., induction vs. inhibition), by increasing the range of the possible choices on each trial. A wider and more abstract set of rules is used, which places greater stress on the inductive component. Furthermore, in contrast to the WCST, the rule that is currently in operation cannot be triggered by a perceptually salient aspect of the stimuli.

Four groups of subjects (one control group and three neurological lesion groups) were used as an initial standardisation sample for the Brixton Test (Burgess & Shallice, 1997). The lesion groups were divided into anterior (lesion involving the frontal lobes), posterior (lesions elsewhere in the cortex not involving the frontal lobes), and bifrontal (bilateral frontal lobe lesions with no posterior involvement). The control participants consisted of healthy volunteers who had no previous history of neurologic or psychiatric disorder, epilepsy, or drug or alcohol abuse problems. The authors found that the unilateral anterior subjects were significantly poorer than the posteriors or the controls, with the posteriors not significantly different from the controls. The bifrontal group achieved the lowest score, however the contrast with the unilateral anterior group failed to reach significance. The split-half reliability of the Brixton Test for the control group was found to be 0.62 ($p<.001$), and overall test-retest reliability to be 0.71 ($p<.001$).

5.3 Executive functioning and aggression – a review of previous research

Although the causes of interpersonal aggression are multifactorial, research suggests an association between low executive functioning and aggressive behaviour (see Giancola, 1995; Moffitt, 1993 for reviews). Individuals with disorders

characterised by antisocial behaviour such as APD (Dolan & Park, 2002), substance use disorders (Giancola & Moss, 1998), CD (Giancola & Mezzich, 2000; Moffitt, 1993) and attention deficit hyperactivity disorder (ADHD: Barkley, 1997) have all demonstrated poor performance on neuropsychological tests of executive functioning. However, although executive functioning has been linked to disinhibited and antisocial behaviour more broadly, only a few studies have examined its relationship to aggressive behaviour more specifically.

The study of clinical populations characterised by aggression has supported the importance of executive functioning in the mediation of aggressive behaviour. While there is little evidence that psychopathy is associated with impaired executive functions (see Blair & Frith, 2000 for review), there have been reports of impairments in antisocial groups more broadly defined (e.g., Dinn & Harris, 2000). A recent review concluded that executive function is impaired in APD, with an estimated effect size of .62 (Morgan & Lilienfeld, 2000).

More precise information about the nature of neuropsychological function in antisocial groups is provided by Dolan and Park (2002). This study involved offenders specifically diagnosed with DSM-IV APD (American Psychiatric Association, 1994) but not other Axis II disorders who had been screened for current Axis I disorders, previous drug or alcohol dependence, learning disability, medication and neurological damage. The tasks were selected as being putatively associated with both dorsolateral and ventromedial frontal lobe function, and included the Tower of London planning test (Owen, Downes, Sahakian, Polkey & Robbins, 1990) and a visual discrimination learning paradigm that tests the ability to shift an attentional set on the basis of feedback information (Owen, Roberts, Polkey, Sahakian & Robbins, 1991). The battery also included a Go/No-Go task. The inhibitory aspects of such

tasks depend on inferior portions of the frontal lobes (Aron, Fletcher, Bullmore, Sahakian & Robbins, 2003). Comparison with non-offender controls matched for age and IQ demonstrated that the APD offenders were impaired at formulating sequences of actions to solve problems on the Tower of London planning test, and that they were impaired at shifting a learnt attentional bias in visual discrimination learning. These two deficits are consistent with the proposal that APD involves dysfunction of circuitry encompassing the dorsolateral prefrontal cortex. In addition, on the Go/No-Go task, the offenders made consistently more errors of commission, suggesting dysfunction in more inferior frontal areas. Further to such findings, CD adolescent males (Lueger & Gill, 1990) and females (Giancola & Mezzich, 2000) have also been shown to perform poorly on neuropsychological tests of executive functioning.

Further experimental studies have provided additional evidence supporting the aggression-executive function relationship. Aggression has been linked to low scores on tests of executive functioning in samples of boys (Giancola et al., 1996; Seguin, Pihl, Harden & Tremblay, 1995), adolescent girls (Giancola, Mezzich & Tarter, 1998a), adult men (Giancola & Zeichner, 1994; Hoaken, Assaad & Pihl, 1998; Lau & Pihl, 1996; Lau, Pihl & Peterson, 1995) and adult women (Hoaken et al., unpublished observations, as cited in Hoaken, Shaughnessy & Pihl, 2003). Foster, Hillbrand and Silverstein (1993) were also able to predict future aggression using neuropsychological measures in a sample of men who had previously committed violent crimes.

5.4 Executive functioning and impulsive- and premeditated-aggression

While there is a large body of literature reporting neuropsychological correlates of violence and aggression, there are few authors in the aggression

literature who have reported deficiencies associated specifically with either impulsive- or premeditated-aggressive individuals. For example, some studies have found that the reduced frontal functioning is present in patients presenting with impulsive-aggression, but not premeditated-aggression (see Blair, 2004 for review).

Using the SHAPS, Dolan and Anderson (2002) grouped male personality disordered offenders into high and low impulsive aggressors. They found a negative correlation between impulsivity and aggression with executive function. Similarly, problems in executive functioning were also found to be a predictor of reactive aggression in adolescent boys at risk for substance abuse (Giancola et al., 1996).

Similar results have been found in other samples utilising a similar classification system as those above. For example, Stanford et al. (1997) found that impulsive-aggressive college students exhibited executive control deficits in the areas of verbal strategic processing and impulse control. Barratt et al. (1997b) also documented an inverse relationship between impulsive-aggression and verbal skills in impulsive-aggressive inmates compared with non-impulsive (premeditated) aggressive inmates and non-aggressive controls. Villemarette-Pittman et al. (2002) showed that the verbal deficits observed in impulsive aggressive college students varied according to the degree of executive demands of the task. Consequently, they concluded that the poorer scores were a result of executive dysfunction and not solely a problem in verbal ability.

Broomhall (2005), in his investigation of reactive (i.e., impulsive) versus instrumental (i.e., premeditated) violent offenders, found that the primarily reactive group was significantly impaired on tasks that involved higher-order executive functions such as verbal inhibition, maintenance of set, cognitive flexibility and the ability to see future consequences. The primarily instrumental group, on the other

hand, were largely intact on executive measures. Similarly Brower and Price (2001) suggest that prefrontal network dysfunction seems to be most specifically associated with a recurrent, impulsive subtype of aggression.

However, while a number of studies have attempted to delineate the abnormal clinical and neurological features that characterise impulsive-aggressive behaviour (Coccaro, 1989; Houston & Stanford, 2001; Stanford et al., 2001), few investigations have attempted to document those features that are clinically relevant to individuals that engage in premeditated aggressive acts. The only paper comparing premeditated-aggressive individuals to non-aggressive controls reports no difference on a variety of neuropsychological tests, except for a single subscale of the WCST, in which the premeditated group exhibited greater failure to maintain set than controls (Stanford et al., 2003b).

The other studies to have investigated premeditated-aggression suffer from a number of methodological limitations. Firstly, the majority of studies have utilised offender samples (e.g., Broomhall, 2005). This population is associated with a number of potential confounding variables (e.g., low IQ) making generalisations of findings to a psychiatric or community sample tentative. Secondly, much of the aggression literature implies some link between psychopathy and aggression (Hare, 1993). While individuals who meet criteria for psychopathy may indeed engage in overt aggressive behaviour (premeditated or impulsive), it is not required or necessarily even a common characteristic of the classification. Therefore, accurate study requires explicit identification of the type and degree of aggressive behaviour exhibited.

In summary, neuropsychological assessment has shown a clear link between impulsive-aggressive behaviour and problems in executive functioning, while few if any cognitive deficits have been demonstrated in premeditated aggressive individuals.

Accordingly, a recent review of frontal lobe dysfunction in violent and criminal behaviour recommends that neuropsychiatric evaluations of violent patients should include neuropsychological assessment of executive function "particularly in cases involving recurrent, impulsive aggression" (Brower & Price, 2001, p. 725).

5.5 The relationship between executive functioning deficits and aggression

The relationship between poor executive function and aggression, according to contemporary theories, may be related to poor strategy formulation, cognitive inflexibility, impulsiveness, and deficiencies in generating alternate non-aggressive socially appropriate responses in provocative situations (Dolan & Anderson, 2002). For example, although patients with frontal lobe damage are frequently aware that their aggressive reactions are inappropriate, they have an impaired ability to self-modulate emotions and behaviours in accord with internal need states and the exigencies of the outside world (Golden et al., 1996).

To formulate a general plan, the categorisation of a series of actions is central to achieving the objective, and clinical studies have implicated the prefrontal cortex in such processes. An aspect of behavioural problems observed in patients with frontal lobe lesions is a failure to achieve an objective of goal-oriented behaviour through a series of simple actions (Burgess & Shallice, 1966). Frontal patients also have difficulty arranging a set of simple open-ended tasks in an appropriate temporal order to achieve a behavioural goal (Shallice & Burgess, 1991). Clinical reports further suggest that patients with prefrontal lesions show impairment when formulating a coherent and structured action plan (Sirigu et al., 1995; Zalla, Plassiart, Pillon, Grafman & Sirigu, 2001).

Ineffectual hypothesis generation, concept formation, and set shifting skills along with poor judgement may also undermine one's ability to generate alternative behavioural responses, and to engage in them, in provocative situations. Inadequate planning, organisation, and temporal ordering skills may further compromise one's ability to correctly execute a series of responses in the proper sequence and manner in order to avoid an aggressive interaction. Finally, compromised behavioural inhibition may allow hostile cognitions and affective states to manifest themselves as overt aggressive/violent acts (Giancola, 1995).

Thus, it can be suggested that the dorsolateral prefrontal cortex plays an important role in the mediation of aggressive behaviour. The dorsolateral region of the prefrontal cortex has been shown to be the neural substrate that subserves the executive cognitive functions, which involve the self-regulation of goal-directed behaviour. Environmental determinants may also play a strong role in the expression of aggression, that is, aggressive behaviour usually occurs in the context of a provocative environment (Murdoch, Pihl & Ross, 1990). As such, Giancola (1995) concludes that the combined effects of dorsolateral prefrontal cortical deficits and a provocative/frustrating environment contribute to the propensity for aggressive behaviour. That is, deficient self and social monitoring, abstract reasoning, and attention skills may compromise one's ability to read and correctly interpret potentially ambiguous social cues which can conceivably lead to misunderstandings and possibly aggression in conflict situations.

A further explanation for the relationship between executive functioning and aggression has been related to the concept of impulsivity. More specifically, it has been hypothesised that aggressive, low-executive functioning individuals are less able to inhibit impulsive behaviours (Lau et al., 1995). Lau and Pihl (1996) tested this

hypothesis by examining whether a monetary incentive could decrease aggressive responding in males. Individuals with low executive functioning (unlike those with high executive functioning) were unable to inhibit aggressive responding in the presence of monetary reward. The authors suggested that this inability might be due to a failure to use inhibitory feedback cues to regulate behaviour.

5.6 Limitations of other studies

While research has documented a relationship between aggressive behaviour and executive functioning deficits, such studies do suffer from some methodological shortcomings. Firstly, much research has focused on conduct problems, which may or may not have included aggression. Secondly, they may not have used a comprehensive assessment of executive function with well-validated tests. Executive function is a complex construct involving several dissociable abilities (Robbins, Weinberger, Taylor & Morris, 1996) and using one test of a component of executive function cannot be claimed to be a test of the entire construct. Therefore, it is necessary to administer a number of different neuropsychological tests of executive functions. Given these factors, there may have been a lack of sensitivity and specificity of measurement of both aggression and executive function in several studies that have failed to find the expected relationships.

A further limitation of previous studies surrounds the issue of comorbidity. Many of the disorders investigated in previous studies on antisocial behaviour feature an „impulsive' component such as drug abuse, alcohol intoxication and abuse, and pathological gambling. Antisocial behaviour can also be comorbid with other mental illnesses such as bipolar disorders, schizophrenia, and ADHD (American Psychiatric Association, 2000). While these conditions in themselves are not necessarily risk

factors for aggressive behaviour, poor frontal lobe function has nonetheless been implicated in all of these conditions. It is important to note, however, that some of the demonstrated dysfunction may be dominantly orbitofrontal rather than dorsolateral.

As noted previously, a number of studies examining participants without significant psychopathology have found some evidence for a relationship between subclinical impairment in executive functioning and antisocial and violent behaviour (Giancola & Zeichner, 1994; Lau et al., 1995; Moffitt & Henry, 1989; Seguin et al., 1995; Stanford et al., 1997), providing an empirical foundation for research on the epidemiology of impaired executive functioning as a risk factor for aggressive behaviour in the general population. Unfortunately, however, most research on the aggression-executive functioning relationship has been limited to clinical, incarcerated, and other small non-representative samples (Hawkins & Trobst, 2000; Morgan & Lilienfeld, 2000). Such research does not take into account those individuals who behave aggressively yet have not come into contact with the criminal justice or mental health systems.

As executive functioning is involved in the planning, initiation, and regulation of goal-directed behaviour (Milner, 1995), deficits in its function often contribute to poor behavioural self-regulation, social skills, and judgement. A „clinical impairment' in executive functioning typically results from damage, often due to injury, in the frontal lobes of the brain. „Subclinical impairment' in executive functioning, on the other hand, is not readily observable or easily diagnosable and may be affected by a range of hereditary, behavioural, and environmental factors (Paschall & Fishbein, 2002). In a review of frontal lobe damage and antisocial behaviour, Kandel and Freed (1989) argued that although hard neurological signs may not be evident, minimal brain dysfunction can still influence behaviour. It is thus imperative to investigate

whether previously demonstrated executive deficits are present in individuals who are functioning ‚normally' by societal standards. This being said, it is acknowledged that it is difficult to ascertain from questionnaire responses the exact level of clinical impairment evident in the experimental groups without concurrent behavioural evidence.

5.7 Aim and hypotheses

There is ample evidence that individuals with antisocial behaviour show impaired performance on measures of executive functioning. However, it should be noted that the frontal lobe positions have been relatively underspecified, typically, they do not distinguish between different forms of executive dysfunction or different regions of the prefrontal cortex. In addition, it remains unclear whether executive dysfunction relates to antisocial behaviour more broadly or to other characteristics, such as aggression, which may contribute to antisocial behaviour. Therefore, the purpose of the current study is to examine functional neuropsychological deficits (i.e., deficits with no identified organic aetiology such as head injury), namely executive functions, in impulsive-aggressive and premeditated-aggressive individuals, as well as controls. A number of well-validated tests of executive function were chosen to assess the performance of the groups across a range of executive abilities.

While previous studies on aggression have demonstrated prefrontal deficits more broadly, it is the aim of the current study to focus on the separable role of the dorsolateral prefrontal system to determine if deficits in this region are related to impulsive-aggression and/or premeditated aggression. As previously mentioned, the orbital region of the prefrontal cortex has been found to be related to emotional regulation and impulsivity, thereby increasing susceptibility to aggressive impulses,

whereas the dorsolateral region of the prefrontal cortex has been found to be primarily responsible for aggressive behaviour due to impairments in executive functioning (Giancola, 1995). The focus of this study is thus on the role of the dorsolateral prefrontal cortex through executive functions in the expression of impulsive- and premeditated-aggression.

Based on the research outlined above, it is hypothesised that in comparison to controls, executive functioning deficits (e.g., planning, problem-solving, inhibition, cognitive flexibility, set-shifting) will be present in impulsive-aggressive individuals, given their hostile impulsive reaction in response to provocation which is demonstrated without forethought or planning (Giancola, 2000). This is in contrast to premeditated-aggression, characterised primarily by planned and controlled aggressive actions directed at attaining a particular goal. By definition, premeditated-aggression is characterised by intact planning abilities and the cognitive regulation of behaviour. It is therefore expected that premeditated-aggressive individuals will not demonstrate executive functioning deficits in comparison to controls.

5.8 Method

5.8.1 Participants

An initial pool of 484 students from the University of Tasmania was screened using the Aggression Questionnaire – Short Form (BPAQ-SF: Bryant & Smith, 2001). Aggression scores were non-normally distributed, with skewness of .796 (*SE* = .115) and kurtosis of .375 (*SE* = .23).

Based on questionnaire responses, 100 participants (female = 68, male = 32) were selected and placed into one of two groups; aggressive (*n* = 70), and non-aggressive controls (*n* = 30). The group selection criterion was one standard deviation

(*SD* = 7.43) above the mean (*M* = 24.14) on the BPAQ-SF for the aggressive group (BPAQ-SF = 31) and one standard deviation below the mean for the control group (BPAQ-SF = 17). The aggression scores differed significantly between the aggressive group (*M* = 37.59, *SD* = 5.75) and control group (*M* = 15.2, *SD* = 1.73), F(1, 99) = 435.93, MSE = 10523.52, $p<.001$, $\eta^2 = .816$.

The aggressive group was further divided into predominantly impulsive-aggressive and predominantly premeditated-aggressive using the Impulsive-Premeditated Aggression Scale (IPAS; Stanford, Houston, Mathias, Villemarette-Pittman, Helfritz & Conklin, 2003a). The IPAS was developed with 15 items related to impulsive-aggression and 15 items to premeditated-aggression. However, based on the original factor analysis, it is unclear which items specifically refer to each subtype as the item principal components analysis identified three factors (impulsive aggression, premeditated aggression, familiarity with target/remorse/agitation), rather than the proposed two. Consequently, a series of items were selected which had clear face validity as reflecting the theoretical constructs of impulsive- and premeditated-aggression. From these, only those that shared variance to form clear impulsive-aggressive and premeditated-aggressive in the four factor analyses (Haden, Scarpa & Stanford, 2008; Kockler, Stanford, Nelson, Meloy & Sanford, 2006; Mathias et al., 2007, Stanford et al., 2003a) conducted on the scale to date were selected, and only if they obtained loadings greater than 0.4 on three of the four analyses. This identified eight impulsive and nine premeditated items (see Table 5.1).

Table 5.1
The Impulsive-Premeditated Aggression Scale (Stanford et al., 2003a)

Impulsive Items

1. I became agitated or emotionally upset prior to the acts*
2. I feel I lost control of my temper during the acts*
3. I consider the acts to have been impulsive*
4. When angry I reacted without thinking*
5. I usually can't recall the details of the incidents well*
6. I feel some of the incidents went too far*
7. My behaviour was too extreme for the level of provocation*
8. I was in a bad mood the day of the incident*
9. I feel I acted out aggressively more than the average person during the last 6 months
10. I knew most of the persons involved in the incidents
11. I typically felt guilty after the aggressive acts
12. I was concerned for my safety during the acts
13. I was in control during the aggressive act
14. I understood the consequences of the acts before I acted
15. I was confused during the acts.

Premeditated Items

1. I think the other person deserved what happened to them during some of the incidents*
2. I am glad some of the incidents occurred*
3. Some of the acts were attempts at revenge*
4. I feel my actions were necessary to get what I wanted*
5. I felt my outbursts were justified*
6. I wanted some of the incidents to occur*
7. I planned when and where my anger was expressed*
8. Prior to the incidents, I knew an altercation was going to occur*
9. Sometimes I purposely delayed the acts until a later time*
10. The act led to power over others or improved social status for me
11. I was under the influence of alcohol or other drugs during the acts
12. Anything could have set me off prior to the incident
13. I felt pressure from others to commit the acts
14. The acts were a „release' and I felt better afterwards
15. My aggressive outbursts were usually directed at a specific person

* Items from the IPAS used in the grouping of the aggression groups.

Participants were placed into either predominantly impulsive-aggressive or predominantly premeditated-aggressive if there was a difference of 25% or greater between their scores on the two subscales, based on the methods of Andreasen and Olsen (1982) and Slaghuis and Bakker (1995). This method led to the exclusion of 15 participants from the 70 aggressive individuals initially identified. The final sample consisted of 38 impulsive-aggressive, 17 premeditated-aggressive and 30 control participants. The number of males and females in each group is shown in Table 5.2.

Table 5.2

Number of males and females in the three participant groups and total sample

	Impulsive-Aggressive	Premeditated-Aggressive	Control	Total
Males	9	11	9	29
Females	29	6	21	56

Mean aggression scores on the BPAQ-SF for the three participant groups are shown in Table 5.3. There was a significant difference in aggression scores between the three groups, $F(2, 84) = 189.68$, $MSE = 4782.43$, $p<.001$, $\eta^2 = 822$. Post hoc Tukey tests indicated that the impulsive-aggressive and premeditated-aggressive groups had significantly higher aggression scores than the control group (ps $< .05$). There were no significant differences in BPAQ-SF scores between the males and females in the impulsive-aggressive group, $F(1, 37) = .33$, $MSE = 9.94$, $p = .57$, $\eta^2 = .009$, premeditated-aggressive group, $F(1, 16) = .2$, $MSE = 10.59$, $p = .66$, $\eta^2 = .013$, or total sample, $F(1, 84) = .71$, $MSE = 98.75$, $p = .4$, $\eta^2 = .008$, however in the control

group, males had significantly higher aggression scores than females, $F(1, 29) = 10.3$, $MSE = 31.56$, $p = .003$, $\eta^2 = .269$.

Participants' ages ranged from 17 to 30 years with a mean age of 19.89 years ($SD = 2.56$). Mean ages for each participant group are presented in Table 5.3. There was no significant difference in age between the three participant groups, $F(2, 84) = .15$, $MSE = .98$, $p = .86$, $\eta^2 = .004$.

Inclusion into this study required all participants to be between 17 and 30 years of age, speak English as their first language and have no history of neurological conditions or head injury. Head injury was defined as being knocked unconscious for any period of time, having been diagnosed with concussion by a physician, or having suffered trauma to the head severe enough to require medical attention. Participants were also excluded if they scored lower than 8 (scaled score) on the Wechsler Adult Intelligence Scale – Third Edition (WAIS-III; Wechsler, 1997) Vocabulary and Digit Span subtests which were completed during the testing sessions to rule out a general cognitive or memory deficit.

Table 5.3

Mean (and standard deviation) scores on the Aggression Questionnaire – Short Form and ages for the three participant groups and total sample

	Impulsive-Aggressive	Premeditated-Aggressive	Control	Total
BPAQ-Revised	37.47 (5.47)	38.18 (7.23)	15.50 (2.08)	29.86 (11.77)
Males	36.56 (4.25)	38.82 (6.97)	17.00 (1.12)	31.34 (10.94)
Females	37.76 (5.83)	37.17 (8.06)	14.76 (1.95)	29.07 (12.24)
Age	19.87 (2.71)	19.95 (1.97)	20.07 (2.72)	19.89 (2.56)

5.8.2 Materials

5.8.2.1 Questionnaires

Aggression Questionnaire – Short Form (BPAQ-SF: Bryant & Smith, 2001). For the purpose of selecting participants who were characteristically aggressive, the BPAQ-SF was selected. The BPAQ-SF, a condensed version of the original 29-item Buss-Perry Aggression Questionnaire (BPAQ; Buss & Perry, 1992), is a 12-item self-report measure that records aggressive feelings and behaviours. Items on both forms of the questionnaire load onto four dimensions of aggression: physical aggression, verbal aggression, anger and hostility. Buss and Perry propose that each subscale represents a different element of aggression; the physical and verbal aggression subscales reflect the instrumental or motor behavioural elements of aggression, the hostility subscale reflects the cognitive elements of aggression, while anger reflects the emotional or affective elements and connects the cognitive and behavioural elements.

The BPAQ-SF includes 12 items from the BPAQ, assessed using a 5-point Likert scale, ranging from 1 (very unlike me) to 5 (very like me). In a study comparing the two measures, Bryant and Smith (2001) found comparable internal consistency (alpha coefficients) among the factors on the measures (Physical Aggression, .79, .80; Verbal Aggression, .83, .80; Anger, .76, .76; and Hostility, .75, .70). The BPAQ-SF shows similar factor loadings for males and females (Bryant & Smith, 2001). Diamond, Wang and Buffington-Vollum (2005) further investigated the BPAQ-SF with a sample of male offenders incarcerated in a psychiatric prison hospital. The four-factor structure was confirmed with this sample; however, exchanging one item on the Bryant and Smith Anger scale, “I flare up quickly and get over it quickly”, with “Sometimes I feel like a powder keg about to explode”

improved the fit of the model and increased the reliability of that scale. Reliabilities were comparable to the original form of the BPAQ when corrected for attenuation.

The authors of the original BPAQ (Buss & Perry, 1992) report strong internal consistency for each of the four subscales and total aggression scores, with alphas ranging from .72 (verbal aggression) to .89 (full score). Test-retest correlations were also sound, ranging from .72 to .80, indicating stability over time. Correlations between self-report and peer nomination were significant, indicating strong reliabilities for individuals to self-report their aggressive traits. These findings have been replicated in many studies, finding internal consistency and stability over time for the measure (e.g., Garcia-Leon et al., 2002; Harris, 1997; Williams, Boyd, Cascardi & Poythress, 1996). Further factor analyses have found similar scale structure to that originally claimed for the questionnaire (Harris, 1997).

The Impulsive-Premeditated Aggression Scale (IPAS: Stanford et al., 2003a). The IPAS is a 30-item, self-report questionnaire that classifies an individual's aggressive behaviour as predominantly impulsive or predominantly premeditated in nature. Participants are asked to consider their aggressive acts during the last six months and complete the IPAS in relation to those acts. Of the 30 items, 15 items focus on impulsive aggression characteristics such as "when angry I reacted without thinking", and 15 items focus on premeditated aggression characteristics, such as "I planned when and where my anger was expressed". The items are scored on a 5-point Likert scale (5 = strongly agree, 4 = agree, 3 = neutral, 2 = disagree, 1 = strongly disagree). The content of the scales are based on previous scales and studies that have attempted to differentiate impulsive and premeditated aggressive participants (Barratt et al., 1999; Barratt et al., 1997b; Heilbrun et al., 1978; Heilbrun, Knopf & Bruner,

1976; Linnoila et al., 1983; Stanford, Greve & Dickens, 1995; Stanford et al., 1997; Virkkunen, De Jong, Bartko & Linnoila, 1989b; Vitiello et al., 1990).

Stanford et al. (2003a), in their study of men referred for aggression problems, found that that the IPAS demonstrated adequate reliability coefficients (Cronbach's αs = .77 for the impulsive scale and .82 for the premeditated scale). The impulsive and premeditated scales were not significantly correlated (r = -.02). The authors also reported strong construct and concurrent validity for the IPAS scales. These findings have been replicated in later studies, finding adequate internal consistency in a forensic sample (impulsive α = .72; premeditated α = .81) (Kockler et al., 2006) and in treated opiate-dependent individuals (impulsive α = 74; premeditated α = .75) (Conner, Houston, Sworts & Meldrum, 2007). Test-retest analyses also indicate good stability, with impulsive and premeditated correlation coefficients .63 and .70 respectively. Also consistent with prior studies of the IPAS, the impulsive and premeditated scales were found to be poorly correlated, supporting the conception that these scales assess different types of aggression (Conner et al., 2007).

The I7 Impulsivity Questionnaire (Eysenck, Pearson, Easting & Allsopp, 1985). The I7 Impulsivity Questionnaire is a 54-item self-report measure which consists of three subscales: impulsiveness, venturesomeness and empathy. High reliability is reported for each of the subscales for both males and females, with alpha coefficients for impulsivity and venturesomeness reported to be around .80 and empathy at .69. A moderate correlation was found between impulsiveness and venturesomeness (r = .38), reflecting the association between the two subscales in terms of a combined impulsivity trait. The authors conclude that this self-report measure robustly assesses three important characteristics in impulsivity research: impulsiveness, venturesomeness, and empathy. It is the inclusion of empathy that

makes this scale of further relevance for the current investigation into aggression, as lack of empathy can be a defining characteristic of premeditated aggression (Meloy, 1997).

5.8.2.2 Executive function measures

The Delis-Kaplan Executive Function System (D-KEFS; Delis, Kaplan & Kramer, 2001). The D-KEFS is a set of nine standardised tests for assessing executive functions in children and adults. The tests measure a range of verbal and non-verbal executive functions, with each test designed to stand alone or be administered with others from the battery. These tests are predominantly an updated version of commonly used stand-alone tests of executive functioning with better standardisation and quantitative error scoring. Each of the nine tests were standardised on over 1700 children and adults aged nine to 89 years. Two subtests from the D-KEFS were chosen for the current study; Verbal Fluency Test and the Trail Making Test.

D-KEFS Verbal Fluency Test. The D-KEFS Verbal Fluency Test is modelled on the Controlled Oral Word Association Test (COWAT) which was developed by Benton and his colleagues (Benton & Hamsher, 1976; Spreen & Benton, 1969). The Verbal Fluency Test is comprised of three conditions; letter fluency, category fluency and category switching. Only letter fluency was chosen for the current study given its sensitivity to frontal involvement (Benton, 1968). In this task, the participant is asked to name in 60 seconds as many words as they can that begin with a specified letter over three trials (F, then S, then A). The participant is constrained by two rules: words cannot include the names of people, places, or numbers, and they cannot provide grammatical variants of previous responses (e.g., take, and then taking, takes).

D-KEFS Trail Making Test. The D-KEFS Trail Making Test involves a series of five conditions: visual scanning, number sequencing, letter sequencing, number-letter switching and motor speed. In all five conditions the stimuli are spread over an 11 x 17-inch area, which provides longer trails and more interference stimuli than the traditional Trail Making Test (Delis et al., 2001). In the Visual Scanning condition, participants are required to cross out all the 3s that appear on the response sheet, which are mixed among a collection of other numbers and letters. In the Number Sequencing condition, participants draw a line connecting the numbers 1-16 in order, with distracter letters appearing on the same page. The Letter Sequencing condition requires participants to connect the letters A through P, with distracter numbers present on the same page. In the Number-Letter Switching condition, participants switch back and forth between connecting numbers and letters (i.e., 1, A, 2, B, etc., to 16, P). Finally, a Motor Speed condition is administered in which participants trace over a dotted line connecting circles on the page as quickly as possible, in order to gauge their motor drawing speed.

Each condition is preceded by a short practice trial. In all conditions, participants were instructed to work as quickly and as accurately as possible. In all but the Visual Scanning condition, the examiner corrected any mistakes by placing an "X" over a wrong connection and participants were asked to continue from the last correct connection. The stopwatch remained running during such corrections. Success at the task is measured by time to completion for each condition.

The D-KEFS Trail Making Test was modelled after the traditional Trail Making Test (Reitan & Wolfson, 1985; see review of test history in Delis et al., 2001), with some important modifications in order to address some limitations of the traditional test. For example, the D-KEFS Trail Making Test contains both number

sequencing and letter sequencing conditions that are completed prior to the switching condition. In addition, the stimulus pages for the number sequencing and letter sequencing conditions contain both numbers and letters, and taken together, the two conditions match the switching conditions well in terms of the number of stimuli to visually scan and the number of lines that need drawing. The D-KEFS Trail Making Test was also created to isolate set-shifting abilities from other component skills required for the task such as visual scanning and motor speed.

Tower of Hanoi (Goel & Grafman, 1995). The Tower of Hanoi task requires the manipulation of several disks onto three rods in order to recreate a given configuration, across three levels of increasing complexity. There are three constraints on the transformation of the start state into the goal state: (1) only one disk may be moved at a time; (2) any disk not being currently moved must remain on a peg; and (3) a larger disk may not be placed on a smaller disk.

The Tower of Hanoi task is widely used as an experimental and diagnostic tool in the neuropsychological literature to gauge planning and problem-solving abilities (Grafman et al., 1992; Shallice, 1982, 1990; Spitz, Minsky & Bessellieu, 1985). The rational underlying this interpretation seems to be that, to successfully complete the task, participants need to "look ahead" several levels and solve the problem in their heads, before physically moving any disks. If they are unable to solve the problem, it follows they were incapable of searching through the moves in their heads, and therefore they are argued to have a "planning" or "look ahead" deficit (Goel, Pullara, & Grafman, 2001).

The Tower of Hanoi task was presented on a Pentium 90 computer with a 17” monitor. Participants used a keyboard to indicate their responses.

Stroop Colour-Word Interference Task (Stroop, 1935). The Stroop task measures freedom from distractibility, selective attention, ability to resolve response conflict, and response inhibition (MacLeod, 1991; Perret, 1974). While the Stroop task is made up of three conditions, it is the interference condition which is of particular importance in this study as it measures the participant's ability to inhibit a dominant response (i.e., reading the word) while attending to the less salient feature of the stimuli in naming the colour in which the word is printed. This condition involves filtering out distracting irrelevant information that can compete with the appropriate response, with the target and distractor being different attributes of the same stimulus. The task therefore also places great demand on selective attention in order to filter out the distracting information. In this task, lower number of errors (the dependent variable) indicates superior inhibitory ability.

The Stroop task was presented on a Pentium 90 computer with a 17" monitor and participants used a keyboard to indicate their responses.

The Brixton Test (Burgess & Shallice, 1997). The Brixton Test consists of a 56-page stimulus booklet. Each page displays the same basic array of ten circles set in two rows of five, with each circle numbered from one to ten. On each page, one of the circles is filled with a blue colour, with the position of this filled circle changing (on most occasions) from one page to the next. The changes in position are governed by a series of simple rules that vary without warning. The participant is shown one page at a time and is asked to decide where the next filled position will be, basing the selection on an apparent pattern or rule derived from the previous pages. The blue circle moves on each succeeding card following seven rules of five different kinds. Participants' answer to the first item is disregarded as it is always a guess. On trials in which there is a rule change, the correct answer is not the actual position where the

blue circle goes next, but where the blue circle would go if the rule change had not occurred.

Using the method described in the test manual, the total errors were recorded and these errors (maximum 54) were converted to scaled scores (e.g., 0 to 7 raw errors were converted to a scaled score of 10, which is classified as „very superior' performance). An overall standardised scaled score based on a scale ranging from 1 (impaired) to 10 (very superior) was used for analysis.

5.8.2.3 Wechsler Adult Intelligence Scale – Third Edition (Wechsler, 1997)

Two subtests from the WAIS-III were selected as control measures. The Vocabulary subtest was included to provide an indication of participants' general intellectual functioning given its correlation of .90 with WAIS-III Full Scale IQ (Wechsler, 1997). Although Milner and Petrides (1984) argued that intelligence is independent of executive function, the capacity for understanding and remembering a set of complex instructions and rules involved in some of the executive tasks included in this study could presumably be affected by poor intelligence (Seguin, Boulerice, Harden, Tremblay & Pihl, 1999). Vocabulary measures expressive vocabulary, verbal knowledge and fund of information. For this subtest, participants were required to define words that were orally presented.

Digit Span involves repeating digits in increasing spans in forward and backward orders. This task was used as a measure of verbal working memory and attentional capacity for verbal information. The number of fully correct responses for both forwards and backwards order was used as an estimate of the participant's working memory capacity (higher score indicates better performance) (Wechsler, 1997).

5.8.3 Procedure

The BPAQ-SF was used to recruit participants from undergraduate psychology classes at the University of Tasmania. Following the screening process, select individuals who qualified as aggressive or control participants were invited to participate in the executive functioning tasks. Approval from the Human Research Ethics Committee (Tasmania) Network was obtained before recruitment procedures took place (see Appendix A for approval letter). All participants received course credit or a small payment for their participation.

Informed consent was obtained from all individuals prior to participation (see Appendix B for participant information sheet and Appendix C for consent form). Participants were tested individually in a quiet room in the School of Psychology at the University of Tasmania and completed the tasks in a counterbalanced order. Each task was explained and comprehension was ensured before beginning each task. The experimenter was blind as to the group the participant was assigned to.

Tower of Hanoi: Participants completed a computerised version of the Tower of Hanoi. The task entailed a brown platform mounted with three vertical rods being presented centrally on the screen. The first stage involved three rings (differing in circumference) stacked on the left-most rod (smaller rings were always stacked on top of larger rings). Participants were instructed to reproduce the same stacking configuration of rings on the right-most rod by moving the rings according to three rules outlined above. Three trials were conducted. The first involved three rings, the second involved four rings, and the third trial involved five rings. If participants had not finished the five rings condition within 100 moves, the task was ended. Performance on this task was assessed by the total number of moves taken to complete the task, with low number of moves indicative of efficiency at problem-

solving. The time taken between the manipulation of the first disk and time taken for subsequent execution of the task was also measured, with quicker times signifying better performance.

Stroop Colour-Word Interference Task: A computerised version of the Stroop task was used. Participants were seated in front of a computer monitor, approximately 60cm from the screen. In the first condition (colour naming), participants reported which of four colour stimuli (RED, BLUE, GREEN, YELLOW) appeared on the screen by pressing the corresponding number (1, 2, 3 or 4) on the keyboard to patch of colours which were numbered across the bottom of the screen. In the second condition (colour word naming), participants were presented with colour words and were required to respond to which of four colour words printed on the screen, again by pressing the corresponding key. The first two conditions consisted of two blocks of 30 trials each. For the second condition, of the 60 trials, 40 trials consisted of the colour word being printed in an incongruent colour, while 20 trials consisted of the colour word being printed in the congruent colour.

In the final condition (interference), participants were required to report the colour of which the word was printed in, ignoring what the actual colour word was by pressing the corresponding key. This condition consisted of four blocks of 30 trials. Of the 120 trials, 88 trials consisted of the word being printed in an incongruent colour, while 32 trials consisted of the colour word being printed in the congruent colour. The latter condition of the task measures the participant's ability to inhibit a dominant response (i.e. reading the word) while attending to the less salient aspect of the colour. For each condition, participants were instructed to respond to the stimuli as quickly and accurately as possible by pressing the appropriate key.

After completion of the executive function measures, participants completed the WAIS-III Vocabulary and Digit Span subtests, the BPAQ, I7 and the IPAS. Participants were fully debriefed at completion of the testing session.

5.9 Results

5.9.1 Participants

Mean scores on the BPAQ and I7 Impulsivity Questionnaire (see Table 5.4) were analysed using separate one-way ANOVAs. On the BPAQ, a significant effect was found for the physical aggression, $F(2, 84) = 52.50$, $MSE = 1927.94$, $p<.001$, $\eta^2 = .561$, verbal aggression, $F(2, 84) = 93.06$, $MSE = 795.18$, $p<.001$, $\eta^2 = .694$, hostility, $F(2, 84) = 35.05$, $MSE = 1063.45$, $p<.001$, $\eta^2 = .461$, and anger subscales, $F(2, 84) = 107.72$, $MSE = 1272.71$, $p<.001$, $\eta^2 = .724$, as well as total aggression, $F(2, 84) = 107.35$, $MSE = 17574.93$, $p<.001$, $\eta^2 = .724$. Post hoc Tukey's indicated that the impulsive-aggressive and premeditated-aggressive groups had significantly higher scores than the control group on all subscales (ps<.05).

While there are no widely accepted scores on the BPAQ indicative of clinical significance, the original article by Buss and Perry (1992) reported a mean score of 73 for a college population aged 18-20 which is comparable to the present study. Similarly Smith and Waterman (2004) reported a mean score of 72.13 for their sample of undergraduate students. In comparison, the mean BPAQ score for the violent and non-violent offenders in their study was 85.97 and 78.24 respectively. Thus, the BPAQ scores for the impulsive-aggressive ($M = 88.61$) and premeditated-aggressive ($M = 91.00$) participants in the current sample are comparable to violent offenders.

On the I7 Impulsivity Questionnaire, a significant effect was found for the impulsivity, $F(2, 84) = 19.22$, $MSE = 333.93$, $p<.001$, $\eta^2 = .319$, venturesomeness,

$F(2, 84) = 3.29$, MSE = 56.83, $p = .042$, $\eta^2 = .074$, and combined impulsivity-venturesomeness subscale, $F(2, 84) = 10.17$, MSE = 531.67, $p<.001$, $\eta^2 = .199$. No significant effect was found for empathy, $F(2, 84) = 1.26$, MSE = 14.92, $p = .291$, $\eta^2 = .030$. Post hoc Tukeys indicated that on the impulsivity subscale, the impulsive-aggressive group had significantly higher scores than the other two participant groups, and the premeditated-aggressive group had significantly higher scores than the control group (*p*s<.05). On the venturesomeness subscale, the premeditated-aggressive group had significantly higher scores than the control group (*p*s<.05). On the combined subscale, the impulsive-aggressive and premeditated-aggressive groups had significantly higher scores than the control group.

Table 5.4

Means (and standard deviations) for the Aggression Questionnaire – Full Scale and I7 Impulsivity Questionnaire for the three participant groups

	Impulsive-Aggressive	Premeditated-Aggressive	Control	Total
BPAQ				
Physical Aggression	27.61 (6.82)	27.24 (7.03)	13.40 (4.16)	22.52 (9.04)
Verbal Aggression	17.74 (2.33)	19.18 (3.13)	9.20 (3.44)	15.01 (5.22)
Hostility	22.37 (6.06)	23.35 (6.64)	12.23 (3.82)	18.99 (7.41)
Anger	22.68 (3.16)	23.00 (4.86)	11.33 (2.76)	18.74 (6.47)
Total Aggression	88.61 (13.03)	91.00 (15.20)	46.83 (10.91)	74.34 (24.05)
I7				
Imp-Vent	21.18 (7.15)	20.12 (6.64)	13.50 (7.30)	18.26 (7.98)
Impulsivity	12.21 (3.91)	9.29 (4.41)	5.90 (4.35)	9.40 (4.99)
Venturesomeness	8.97 (4.37)	10.82 (3.52)	7.60 (4.21)	8.86 (4.27)
Empathy	13.81 (3.31)	12.24 (3.88)	13.17 (3.36)	13.27 (3.46)

Eysenck et al. (1985) report means and standard deviations for males and females for each of the subscales of the I7 Impulsivity Questionnaire. In comparison to these means, on the impulsivity and venturesomeness subscales, the impulsive-aggressive and premeditated-aggressive group had higher scores. On the empathy subscale, the impulsive-aggressive and control groups had higher scores.

5.9.2 Executive function measures

For the Trail Making Test and Brixton Test, completion times and number of errors were respectively converted to standard scores as described in their manuals. Success on the Stroop task was measured by the number of errors made in each of the three conditions. Mean scores (see Table 5.5) were analysed using separate one-way ANOVAs (see Table 5.6 for results of ANOVAs).

Tukey post hoc tests were conducted where the ANOVA was significant ($ps<.05$). The results of the analyses revealed that for Trails Switching, the impulsive-aggressive group scored significantly lower than the control group. The premeditated-aggressive group did not differ significantly from the other groups. On Tower of Hanoi three-rings the impulsive-aggressive group took significantly more moves than the control group to complete the task, and on the five-rings condition, the impulsive-aggressive group took significantly more moves than both the premeditated-aggressive and control groups. No significant group differences were found for the 4-rings condition. The impulsive-aggressive group produced significantly fewer words than the premeditated-aggressive and control groups on the Verbal Fluency Test. On the Stroop task, the impulsive-aggressive group made significantly more mistakes than the control group on the interference condition. The premeditated-aggressive group did not differ from either the impulsive-aggressive or control groups.

5.9.3 Wechsler Adult Intelligence Scale – Third Edition

The premeditated-aggressive group scored significantly higher than the impulsive-aggressive group on Vocabulary and Digit Span. The control group did not differ significantly from the impulsive-aggressive or premeditated-aggressive groups on either subtest (all *p*s>.05). Mean scores on the WAIS-III subtests for the three participant groups fell within the ‚average' range. No participant scored below the average range on either subtest.

ANCOVAs were conducted to determine if group differences on the executive function measures remained significant after controlling for the WAIS-III measures. The covariate Vocabulary was not significant for any of the variables, Verbal Fluency, $F(1, 81) = 3.63$, $MSE = 196.22$, $p = .06$, Trails Switching, $F(1, 81) = .33$, $MSE = 1.58$, $p = .568$, Tower of Hanoi 3 rings, $F(1, 81) = .48$, $MSE = 6.12$, $p = .489$, Tower of Hanoi 5 rings, $F(1, 81) = .05$, $MSE = 19.48$, $p = .832$, Stroop Interference, $F(1, 81) = 1.11$, $MSE = 12.76$, $p = .296$. Group differences remained significant after controlling for Vocabulary on Verbal Fluency, $F(2, 81) = 12.2$, $MSE = 658.83$, $p<.001$, Trails Switching, $F(2, 81) = 5.28$, $MSE = 25.49$, $p = .007$, Tower of Hanoi 3 rings, $F(2, 81) = 5.64$, $MSE = 71.47$, $p = .005$, Tower of Hanoi 5 rings, $F(2, 81) = 8.7$, $MSE = 3737.5$, $p<.001$, and Stroop Interference $F(2, 81) = 7.29$, $MSE = 84.1$, $p = .001$.

The covariate Digit Span was not significant for any of the variables, Verbal Fluency, $F(1, 81) = 1.93$, $MSE = 106.58$, $p = .168$, Trails Switching, $F(1, 81) = 1.05$, $MSE = 5.04$, $p = .308$, Tower of Hanoi 3 rings, $F(1, 81) = .02$, $MSE = .22$, $p = .895$, Tower of Hanoi 5 rings, $F(1, 81) = .52$, $MSE = 222.92$, $p = .472$, Stroop Interference, $F(1, 81) = 3.33$, $MSE = 37.39$, $p = .072$. Group differences remained significant after controlling for Digit Span on Verbal Fluency, $F(2, 81) = 12.36$, $MSE = 681.13$,

p<.001, Trails Switching, F(2, 81) = 4.81, MSE = 22.98, p = .011, Tower of Hanoi 3 rings, F(2, 81) = 5.09, MSE = 64.82, p = .008, Tower of Hanoi 5 rings, F(2, 81) = 9.8, MSE = 4187.36, p<.001, Stroop Interference, F(2, 81) = 6.62, MSE = 74.32, p = .002.

Table 5.5

Means (and standard deviations) for the three participant groups on the executive function and WAIS-III measures

	Impulsive-Aggressive	Premeditated-Aggressive	Control	Total
Verbal Fluency	31.53 (6.53)	41.82 (6.04)	40.17 (9.11)	36.64 (8.72)
Trails				
Visual Scanning	11.66 (1.28)	11.94 (1.34)	11.20 (2.17)	11.55 (1.67)
Number Sequencing	11.39 (1.59)	10.94 (1.25)	10.73 (2.36)	11.07 (1.85)
Letter Sequencing	11.71 (1.47)	11.65 (1.22)	11.03 (1.87)	11.46 (1.59)
Switching	8.79 (2.22)	10.06 (2.97)	10.60 (1.54)	9.68 (2.31)
Motor	12.08 (1.15)	11.76 (1.21)	11.60 (1.69)	11.84 (1.38)
Tower of Hanoi				
3 rings moves (no.)	11.39 (4.48)	9.35 (2.64)	8.63 (2.47)	10.01 (3.73)
4 rings moves (no.)	29.45 (14.34)	22.24 (14.23)	22.53 (10.01)	25.56 (13.27)
5 rings moves (no.)	79.21 (21.97)	59.76 (17.20)	59.00 (20.56)	68.19 (22.68)
3 rings time (sec.)	37.36 (23.99)	33.35 (22.23)	30.00 (21.39)	33.94 (22.72)
4 rings time (sec.)	92.26 (61.86)	91.82 (135.84)	75.77 (62.28)	86.35 (81.25)
5 rings time (sec.)	256.58 (134.97)	274.06 (337.71)	201.93 (99.66)	240.79 (184.53)
Stroop (errors)				
Patch	0.55 (0.80)	0.47 (0.87)	0.73 (1.23)	0.60 (0.98)
Colour	1.47 (2.57)	1.23 (1.35)	0.83 (1.29)	1.20 (1.97)
Interference	7.53 (3.87)	5.71 (3.12)	4.17 (2.85)	5.98 (3.68)
Brixton	7.29 (1.75)	7.65 (1.62)	7.63 (1.33)	7.48 (1.58)
WAIS-III				
Vocabulary	10.71 (1.29)	12.47 (2.35)	11.37 (1.79)	11.29 (1.82)
Digit Span	10.05 (1.68)	12.00 (2.57)	11.03 (2.04)	10.78 (2.12)

Table 5.6

Results of ANOVAs for the executive function and WAIS-III measures

	F	MSE	*p*	η^2
Verbal Fluency	16.36	911.79	<.001***	.285
Trails				
Visual Scanning	1.22	3.36	.301	.029
Number Sequencing	1.13	3.85	.329	.027
Letter Sequencing	1.69	4.22	.191	.040
Switching	6.06	28.98	.004**	.129
Motor	1.11	2.10	.335	.026
Tower of Hanoi				
3 rings moves (no.)	5.45	68.53	.006**	.117
4 rings moves (no.)	3.09	518.49	.051	.070
5 rings moves (no.)	9.83	4177.81	<.001***	.193
3 rings time (seconds)	0.87	452.31	.422	.021
4 rings time (seconds)	0.39	2599.1	.680	.009
5 rings time (seconds)	1.08	36792.06	.344	.026
Stroop				
Colour Matching	0.47	0.45	.629	.011
Colour Naming	0.88	3.45	.418	.021
Interference	8.26	95.39	.001**	.168
Interference Time	0.32	27255.27	.724	.008
Brixton	0.51	1.28	.604	.012
WAIS-III				
Vocabulary	6.18	18.32	.003**	.131
Digit Span	5.87	23.66	.004**	.125

* $p<.05$, ** $p<.01$, *** $p<.001$.

5.10 Discussion

This study investigated executive functioning in impulsive- and premeditated-aggressive individuals. While previous research has established a relationship

between neuropsychological dysfunction and aggression (e.g., Houston et al., 2003), the purpose of the current study was to extend this research to delineate the relationship between impulsive- and premeditated-aggression and possible executive deficits. Results support and extend previous findings, demonstrating that impulsive-aggressive individuals perform more poorly than both premeditated-aggressive individuals and controls on measures of executive functioning, including cognitive flexibility, planning, problem-solving, and flexibility of verbal thought processes. Consistent with previous work, premeditated aggressors did not differ significantly from normal controls on these measures (Barratt et al., 1997b; Stanford et al., 2003b).

The current study was also intended to rectify certain methodological limitations of past research distinguishing between measures of dorsolateral and orbitofrontal functioning. The tasks chosen for the current study were predominantly measures of dorsolateral functioning, and thus indicate that deficits in dorsolateral functioning may be a key factor in the expression of impulsive-aggression. Such hypotheses do not hold for premeditated-aggression, however, with such individuals demonstrating no deficits on dorsolateral prefrontal measures.

The findings of dorsolateral cognitive deficits cannot be attributed to a general cognitive impairment in impulsive-aggressive individuals relative to the control group. This is indicated by the fact that on those measures not related to frontal lobe function (such as the four baseline conditions on the Trail Making Test), there was no deficit. In addition, a general intellectual or memory impairment in the impulsive-aggressive group was not demonstrated in their performance on the WAIS-III Vocabulary and Digit Span subtests.

5.10.1 Verbal Fluency Test

On the Verbal Fluency Test, the impulsive-aggressive group produced significantly fewer words than the premeditated-aggressive and control groups, indicating poorer verbal fluency and verbal strategic processing.

Impaired executive function can have a significant effect on the expression of other cognitive modalities, and within the impulsive-aggressive population, verbal impairments and abnormalities of language processing regions of the brain have been demonstrated to be one of the most compromised (Barratt et al., 1997a; Barratt et al., 1997b; New et al., 2002, 2004; van Elst, Woermann, Lemieux, Thompson & Trimble, 2000; Villemarette-Pittman et al., 2002; Woermann et al., 2000). More specifically, it is in the domain of verbal mediation where their deficit may have significant effects on social interactions.

Prefrontal verbal cognitive processes play an important role in the modulation of emotional processes in humans. While the nature of the language processing abnormalities and the connection between language processing and the modulation of impulsive aggression remain to fully elucidated, several hypotheses emerge from this finding. Language processing regions may mediate executive abilities important to the regulation of aggressive impulses, such as deductive reasoning, cognitive restraint of aggression, cognitive modulation of emotion, and/or reflective functioning. Furthermore, deficient modulation of verbal expression may also lead to increased frustration in provocative situations (Miller, Collins & Kent, 2008).

Verbal skill deficits may also contribute to impulsive-aggressive behaviour through an inability to use verbal internal controls to inhibit inappropriate behaviour (Dolan & Anderson, 2002). Language-based mechanisms of self-control range from virtually automatic motor programming for inhibiting simple behaviours (e.g., “No”)

to thinking through the presenting situation and evaluating the range of possible responses before responding in a particular way to that situation.

Such theories were also proposed by Tarter, Hegedus, Winsten and Alterman (1984) and Yeudall (1980) who suggested that verbal deficits prevent children from developing internal verbally-based means of inhibiting antisocial impulses. Mungas (1988) proposed that poor expressive verbal skills preclude more adaptive, verbal mediation of behaviour, especially in emotionally charged situations. One's behaviour can thus suffer from a lack of internal verbal synthesis that would normally precede and guide behaviour (Fuster, 1997). Therefore, as a group, impulsive-aggressive individuals may be vulnerable to poor internal modulation of their behaviours. It may be that they cannot „talk' themselves through alternate solutions or responses, nor can they adequately utilise verbal negotiation in a conflict situation. They may employ mostly unsuccessful problem-solving strategies that further limit their ability to resolve problems.

5.10.2 Trail Making Test

On the Trail Making Test, the impulsive-aggressive group scored significantly lower than the premeditated-aggressive and control groups on the letter-number switching condition. This deficit in the ability to shift set (also known as cognitive flexibility) suggests impairment in the ability to constantly change response given the environmental demands faced by the individual.

This result supports the findings of Stanford et al. (1997) who also reported deficits on the Trail Making Test Part B in a sample of students classified as impulsive-aggressive by self-report. In contrast, however, Stevens, Kaplan and Hesselbrock (2003), in their investigation of executive functioning in APD found no significant group differences on the task between APD men, men who had a previous

diagnosis of CD, and controls. This suggests that a deficit in set-shifting may be related to aggressive behaviour specifically rather than merely the presence of antisocial behaviour which may or may not have an aggressive component.

Mental flexibility is a core component of effective executive functioning. It allows an individual to determine and employ alternative solutions to novel social situations. Cognitive flexibility involves the individual's awareness of possible options and alternatives in any given situation, a willingness to be flexible and adapt to the situation, as well as self-efficacy in being flexible (Chesebro & Martin, 2003).

Poor cognitive flexibility may be a risk factor for aggression through its impact on problem-solving abilities (Lezak et al., 2004). That is, a lack of cognitive flexibility, or mental rigidity, may preclude impulsive-aggressive individuals from generating and employing non-aggressive solutions to confrontational situations. Perseveration on a current course of action, or the lack of ability to switch attention, may lead to dysfunctional problem-solving actions which cause interpersonal conflict. In contrast, for non-aggressive individuals, although aggression is a viable option, it appears that by being cognitively flexible, they are able to access a wide range of options which is sufficient enough to enable them to find more effective ways of dealing with situations (Chesebro & Martin, 2003).

Such hypotheses regarding the effect of cognitive rigidity on aggression can only be associated with the expression of impulsive-aggression, as the premeditated-aggressive group did not present with deficits on the Trail Making Test. This is in line with Pham, Vanderstukken, Philippot and Venderlinder (2003) in their assessment of planning and cognitive flexibility in a group of criminal psychopaths. The results of Pham et al. confirmed their hypothesis that psychopaths would perform similarly to

controls on measures of flexibility. This result would be expected, however, given the premeditation and planning involved in such aggressive acts.

This theory is supported by Deu (1998) in his research on cognitive flexibility and criminality. He reported that those offenders with high cognitive flexibility were more likely to report that they would reoffend as they were better able to devise ways of eluding detection. In line with this, McGuire (2001) found that cognitive rigidity resulted in a lack of consideration of alternatives and consequences.

These results, taken together, suggest that a rigidity of response without considering alternative responses, may contribute to aggressive social responses. With less adept interpersonal problem-solving skills, impulsive-aggressive individuals do not learn to deal with conflicts in a pro-social manner. According to cognitive social learning theory, however, effective problem-solving is not an automatic response, but is a learned skill and as such can be acquired (McGuire, 2001; Hollin, 2001).

5.10.3 Tower of Hanoi

On the Tower of Hanoi three-ring and five-ring conditions, the impulsive-aggressive group took significantly more moves than the premeditated-aggressive and control groups to complete the task, indicating poorer problem-solving and planning abilities in this group. On the four-ring condition, there were no significant differences between the groups.

These findings suggest that impulsive-aggressive individuals have particular difficulties with problems requiring higher level planning ability. Although they are able to solve as many problems as controls (i.e., all participants were able to solve the 3-, 4-, and 5-ring problems), they solved these problems with a significantly higher number of moves. This pattern is similar to that reported in individuals with APD

(Dolan & Park, 2002), frontal lobe patients (Owen et al., 1990) and chronic schizophrenics (Pantelis et al., 1997) using the similar Tower of London task. Furthermore, research suggests that compared with non-aggressive controls, aggressive offenders use a limited range of alternatives to solve interpersonal problems, rely more on verbal and physical aggression, and consider fewer consequences of their actions (Slaby & Guerra, 1988).

The prefrontal cortex engages in temporally oriented programming to accomplish tasks. Deficits in prefrontal functioning, therefore, often result in disorganisation and impaired problem-solving (Hall, 1993; Stuss & Benson, 1984), and a failure to anticipate consequences (Kandel & Freed, 1989). Even when coupled with a normal IQ, these problems may be significant. The ability to plan behaviour is a critical executive function used in everyday life, and involves activities such as thinking ahead, setting goals, determining a course of action, and using logic to proceed through a task or problem. Deficits in the ability to plan may result in inappropriate or self-defeating behaviour and a lessened capacity to self-correct, learn and think flexibly which will be particularly detrimental in situations lacking clear rules and structure. The inability to generate a suitable response may exacerbate frustration and the tendency to reflexive emotional responding (Hawkins & Trobst, 2000).

Although the impulsive-aggressive group did take a significantly greater number of moves to complete at least two of the Tower problems, they did not take a significantly longer period of time to do so. The absence of significantly lengthened thinking times suggests that they impulsively make moves before they have generated an appropriate solution to the problem. This is in contrast to the premeditated-

aggressive and control groups who spent a longer period of time planning their moves in order to complete the task using fewer moves.

Planning on the Tower of Hanoi task also places a significant load on spatial working memory and neuroimaging studies indicate that working memory tasks engage the dorsolateral prefrontal cortex among other areas (Barch et al., 1997; Cohen et al., 1994; Courtney, Ungerleider, Keil & Haxby, 1996; Jonides et al., 1993). However, it is important to note that the impulsive-aggressive group did not differ from controls on the WAIS-III Digit Span subtest, a specific measure of working memory. This suggests that the impaired performance demonstrated by this group on the Tower task could be placed on their lack of planning and "looking ahead" abilities, a functional deficit in neural networks involving the dorsolateral prefrontal cortex.

5.10.4 Stroop Colour-Word Interference Task

Impulsive-aggressive individuals had significantly greater difficulty inhibiting a prepotent response than controls, which could not be accounted for by differences in reaction time on the Stroop Task. Difficulty inhibiting a prepotent response during the Stroop task may indicate dysfunction in the dorsolateral prefrontal cortex, while it could also represent selective attention deficits attributed to the anterior cingulate cortex. Given the non-significant group differences on the two baseline conditions, the result cannot be attributed to a general deficit in colour recognition or colour-word reading.

The inability to inhibit inappropriate or exaggerated responses may have a particularly salient influence in propelling one toward violent responding. This deficit may result in an inability to maintain emotional equilibrium, and an inability to

control the behavioural expression of mood changes (Golden et al., 1996). Grafman et al. (1996) argue that in the normal brain "when schema-like knowledge (which would include rules of behaviour), stored in the frontal lobes, is activated, it leads to an inhibition of more primitive reactions (e.g., violent or aggressive behaviour) to environmental provocation" (p. 1231). They further state that the knowledge stored in the frontal lobes plays an executive role over one's behaviour and takes the form of understanding plans and social rules that have an overall goal rather than simply reacting to environmental provocations or demands as they arise. Hence, if such knowledge contained in the frontal lobes is less accessible due to dysfunction in this region, the inhibitory benefit of such information is diminished and inappropriate behaviours are more likely to emerge. Thus, individuals with frontal lobe impairment may experience heightened and exaggerated emotional responses to events, have difficulty inhibiting these response, and respond in accord with these emotions in an exaggerated or inappropriate fashion.

Interestingly, the premeditated-aggressive group did not differ from either the impulsive-aggressive or control group. This finding is in contrast with the self-report measure of impulsivity, the I7 Impulsivity Questionnaire, in which the impulsive-aggressive group had significantly higher impulsivity scores than the premeditated-aggressive and control group. On the combined impulsivity-venturesomeness scale, however, both aggressive groups had significantly higher scores than the control group. It may be that the higher levels of characteristic venturesomeness lead the premeditated-aggressive groups to higher error rates on this task rather than characteristic impulsivity leading to a failure to inhibit responses. That is, this group may not have been concerned about making a higher number of mistakes on the task

given the lack of negative outcomes. In comparison, the impulsive-aggressive group made significantly higher number of mistakes due to their impulse control deficits.

Executive cognitive deficits may manifest as an impulsive behavioural style, and, in line with this, the impulsive-aggressive individuals scored significantly higher on a self-report measure of impulsive behaviour than did the premeditated-aggressive and control individuals. Executive functioning also involves resistance to interference from automatic responses (Barkley, 1996) and it may be that this cognitive control system is involved in regulating aggression (Eisenberg, Smith, Sadovsky & Spinrad, 2004; Posner & Rothbart, 2000). Some theorists have argued that cognitive control resources must be recruited in order to resolve conflict between two response options (Wilkowski, Robinson & Troop-Gordon, 2010). In tasks such as the Stroop task, participants' responses are slowed considerably when response conflict is first introduced. However, response conflict is immediately registered within the anterior cingulate cortex (Botvinick, Nystrom, Fissell, Carter & Cohen, 1999), leading to the recruitment of cognitive control resources within the dorsolateral prefrontal cortex (Botvinick et al., 2001).

This proposed cognitive control system can be used to regulate aggressive behaviour in that once an individual has recruited this system, they should be more able to inhibit socially inappropriate behaviours (Wilkowski & Robinson, 2010). In support of this proposition, Wilkowski and Robinson (2008) found that individuals reporting low trait anger demonstrated superior abilities in resolving response conflict following the activation of hostile thoughts. Thus in respect to the current findings, it may be that impulsive-aggressive individuals are less able to resolve this response conflict, leading to heightened aggressive responding in social situations.

5.10.5 The Brixton Test

No significant group differences were found on the Brixton Task. While the Brixton Test has not been used in previous studies on aggression, this non-significant finding is inconsistent with previous studies using the WCST (e.g., Dolan & Anderson, 2002). The Brixton Test examines the ability to discern a rule set, adhere to it, and adjust it in the event of a rule change. However, as mentioned previously, the Brixton Test places greater emphasis on the inductive reasoning component of the task through increasing the set of rules used. In the Brixton Test, the rules which have to be attained pertain to the relationship between succeeding stimuli stressing the inductive process, in contrast to the WCST in which the rules directly relate to perceptual features on each card. Furthermore, the stimuli are less prone to automatically trigger over-learned stimulus-response associations and thus are less liable to induce perseverative behaviour (Reverberi, Lavaroni, Gigli, Skrap & Shallice, 2005). Thus, the observed result may be caused by the aggressive individuals not having a specific deficit in inductive reasoning, in contrast to the observed concept attainment impairment as measured by the WCST. Alternatively, as suggested above, the deficit observed on the WCST may be related to antisocial traits more broadly rather than to aggression per se.

A further explanation for this result lies in the specificity of the task to the frontal lobes more broadly in contrast to the predominant dorsolateral involvement in the other executive tasks used in this study. Thus it may be that the impulsive-aggressive individuals have a more focal dysfunction related to this region rather than the frontal lobes as a whole. Further research delineating the specific regions of the brain involved in the Brixton Test is needed to clarify such hypotheses as well as for

broader neuropsychological assessments of individuals with injury to the prefrontal cortex.

A related explanation is that the task demands of the Brixton Test are not sufficiently sensitive to adequately discriminate highly educated subjects. The Brixton Test was standardised using samples of individuals with lesions to various areas of the brain. Significant injury to the frontal lobes would be expected to produce more profound deficits in comparison to individuals with proposed sub-clinical impairment in this region. The overall high cognitive and functional level of these participants suggests that possible frontal deficits may be subtle and merely represent the low end of the normal range of functioning. Subtle deficits, however, can have profound effects on behaviour. Further research is nevertheless required before any reasonable speculation can be advanced.

5.10.6 Personality measures

On the impulsivity-venturesomeness subscale of the I7 Impulsivity Questionnaire, the impulsive-aggressive and premeditated-aggressive groups had significantly higher scores than the control group, and on the venturesomeness subscale, the premeditated-aggressive group had significantly higher scores than the control group. With regard to the findings from both the BPAQ and I7 questionnaires, it is not surprising that individuals displaying aggressive behaviour would show significant personality pathology. Individuals displaying chronic aggression problems, regardless of the type of aggressive behaviour, tend to score higher on these personality constructs (Houston & Stanford, 2001; Mathias & Stanford, 1999; Stanford, Greve, Mathias & Houston, 1998; Stanford et al., 2003b). Villemarette-Pittman et al. (2002) found that impulsive-aggressive college students scored higher

than controls on all personality measures (BPAQ, Lifetime History of Aggression Questionnaire, Barratt Impulsiveness Scale). Similarly, Houston et al. (2003) found that individuals classified as Primary Aggressors (both impulsive and premeditated aggressive) scored significantly higher on measures of aggression, anger, hostility, neuroticism, and lifetime history of aggression than those deemed Secondary Aggressors.

On the impulsivity subscale, the impulsive-aggressive group had significantly higher scores than the premeditated-aggressive and control groups. This finding is not surprising given the recognised impulsive-aggressive behaviour demonstrated by this group. Barratt (1991) suggested that impulsive aggression is related to both the level of impulsiveness or impulse control, and the anger level of the individual. Later, he proposed a more specific explanation of the relationship, proposing that some people are predisposed to responding to certain stimuli or situations with feelings of anger that may lead to an aggressive response (Barratt, 1994). If such a predisposition is combined with a high level of impulsivity, then the difficulty of inhibiting responses that is characteristic of impulsive-aggressive individuals involves a low response control and this facilitates aggressive behaviour. From this viewpoint, impulsivity is related to situational control while anger is related to emotional drive.

On the venturesomeness scale, the premeditated-aggressive group had significantly higher scores than the other two groups. This finding is comparable to that of Alexander, Allen, Brooks, Cole and Campbell (2004) in their study of aggression, self-control and instrumental views. The authors reported that in an offender sample, instrumental beliefs (i.e., the view that aggression is an instrumental act aimed at imposing control) were associated with impulsive risk seeking. Such findings suggest that premeditated aggressors do not demonstrate the normal

avoidance to possible disadvantageous consequences arising from risky situations. Such situations include dangerous recreational activities as indicated in the I7 and can also relate to the demonstration of planned aggressive acts.

On the empathy subscale of the I7 Impulsivity Questionnaire, there was no significant difference found between the three participant groups. This result is surprising given the proposed link between premeditated-aggression and psychopathy, a personality pathology characterised by a lack of empathy and remorse. One potential explanation is that although they do engage in premeditated-aggressive acts, the higher levels of empathy present in this group may prevent them from engaging in more serious aggressive acts which have the potential to cause serious harm to others. Such restraint may have resulted in this population not coming into contact with law enforcement authorities or the recognition of a clinically significant impairment in this sample. In contrast, the levels of empathy present in the impulsive-aggressive group were expected given the identified guilt and remorse used to characterise this group.

5.10.7 Psychopathy and Antisocial Personality Disorder

Studies specifically focusing on dorsolateral prefrontal functioning in psychopathic populations have produced conflicting findings largely due to the variation in diagnostic criteria for psychopathy and differences in the nature of the comparison group (Devonshire, Howard & Sellars, 1988; Gorenstein, 1982; Hare, 1984; Lapierre et al., 1995). Psychopathy assessed using the Hare Psychopathy Checklist – Revised (PCL-R; Hare, 1991) emphasises the interpersonal (callous-unemotional) aspects of antisocial behaviour rather than the behavioural components, such as aggression, which receive more emphasis in the criteria for DSM-IV-TR APD (American Psychiatric Association, 2000) and other measures of psychopathy such as

the SHAPS (Blackburn, 1982). The majority of studies, using the PCL-R report no differences on tests of executive functioning with psychopaths compared with non-psychopaths, suggesting that PCL-R psychopathy may be specifically linked with deficits in orbitofrontal function (Lapierre et al., 1995) and/or amygdala dysfunction (Blair, 2001) rather than dorsolateral prefrontal function. It can be suggested then that aggression, which constitutes a behavioural component of psychopathy, is associated with dorsolateral prefrontal deficits.

APD is a heterogeneous disorder and different aspects of its complex clinical presentation may reflect dysfunction in discrete neural systems. Similar to psychopathy, DSM-IV-TR APD (American Psychiatric Association, 2000) does not require an individual to be aggressive to receive a diagnosis. In a recent study, Crowell, Kieffer, Kugeares and Vanderploeg (2003) found that APD individuals performed at comparable levels to psychiatric and normal controls on measures of executive function. In contrast, Dolan and Anderson (2002) found that subjects with APD displayed impairments on executive tasks. These findings suggest that executive dysfunction may be differentially present in antisocial individuals depending on the aetiology of those antisocial traits and behaviours. Therefore, a more focused analysis on specifically characteristic aggression provides much more detailed insight into the causes of antisocial behaviour more broadly. Such non-significant findings in recent studies of executive functioning in antisocial populations may thus merely be due to the lack of characteristic aggression in the sample.

The results of this study therefore suggest that executive functioning deficits are more likely to be related to the actual behaviour of aggression, rather than to diagnostic labels that are only correlated with aggression. As aggressive behaviour is not a necessary diagnostic feature of any of these externalising psychiatric disorders,

it is possible that participants in the studies that reported negative findings were less aggressive than those in the studies that reported positive findings. This argument is supported by a study showing that, compared with psychiatric patients arrested for non-violent crimes; only those arrested for violent crimes exhibited executive functioning deficits (Krakowski et al., 1997). As outlined previously, further support has been reported in normal young adult men (Giancola & Zeichner, 1994; Hoaken et al., 1998; Lau & Pihl, 1996; Lau et al., 1995), women (Hoaken et al., unpublished observations, as cited in Hoaken et al., 2003), and preadolescent boys (Seguin et al., 1995), preadolescent boys at high risk for a substance use disorder (Giancola et al., 1996), and adolescent girls with a substance use disorder (Giancola, Mezzich & Tarter, 1998b).

5.10.8 The link between executive functioning deficits and impulsive-aggression

These results suggest that inefficient executive functioning, evidenced by deficits in dorsolateral functioning is a contributing factor in the development and maintenance of the antisocial behaviour displayed by impulsive aggressors. It is proposed that such executive dysfunction may interfere with their ability to set and achieve goals, adaptively self-regulate behaviour, and consider the future implications of these behaviours.

Impairment in the ability to change behaviour in response to environmental changes in combination with verbal fluency deficits could produce significant difficulties in social situations. When faced with a social situation involving conflict, impulsive-aggressive individuals may become confused by multiple environmental changes (for example, differing arguments), and provoke or be provoked into aggressive behaviour. In support of this, by the definition used in this research, the

aggression demonstrated by the impulsive-aggressors is defined by a high level of spontaneity, lack of planning and heightened agitation following a provocation or conflict (Houston et al., 2003).

As such, it would appear that impulsive-aggressive individuals, due to possible dorsolateral dysfunction in combination with numerous social and psychosocial influences, display a type of ‚acquired sociopathy', which Damasio et al. (1990) described as reactive, emotionally-driven aggression toward a person related to inhibitory dyscontrol. In comparison, premeditated-aggression, which is associated with Factor 1 psychopathy features, may be related to the temporal, premotor cortices and the amygdala, given its characteristic goal-directed motor behaviour and lack of empathy and remorse. Further research involving more thorough neuroimaging techniques in conjunction with the neuropsychological tests would be required to shed more light on such hypotheses regarding premeditated aggression.

With regard to the way in which low executive functioning contributes to aggressive behaviour, Giancola (1995) has advanced a heuristic model arguing that, given a sufficiently provoking environmental context, compromised executive functioning contributes, in part, to the elicitation of physically aggressive behaviour by reducing behavioural inhibition and the ability to generate non-aggressive alternative forms of behaviour. For example, impaired self-monitoring, abstract reasoning, and attentional skills may impede the ability to correctly interpret potentially ambiguous social cues during interpersonal interactions, which may lead to misattributions in the perception of threat or hostility in conflict situations. In addition, ineffectual hypothesis generation, poor concept formation, and cognitive inflexibility, as identified in the current study, may impair one's ability to generate and implement alternative non-aggressive behavioural responses in hostile

interpersonal situations. Inadequate planning and organisation capacities may further compromise one's ability to correctly executive a series of responses in the appropriate sequence and manner in order to avoid an aggressive interaction. Finally, compromised cognitive control over behaviour may allow aggressive cognitions and affect to manifest as overt aggression.

Knowledge stored in the prefrontal cortex plays a managerial role in the control of behaviour and takes the form of understanding, planning, and understanding social rules. This knowledge enables humans to engage in a series of behaviours that have an overall goal, rather than simply reacting to the moment-by moment provocations or demands of the environment. Within this framework, it would be expected that prefrontal dysfunction would impair the ability to access and sustain such managerial knowledge and would thus bias the regulation and expression of behaviour away from plans, social rules, and mental schemas towards environmental hyper-responsiveness, making impulsive aggression more likely (Grafman et al., 1996).

The ability to defuse a hostile situation, which may involve de-escalating hostility in another individual, is crucial for the inhibition of an aggressive reaction. Without de-escalation, prolongation of the encounter could heighten frustration and anger in the individual, which would lead him or her to a reactive aggressive response. In order to inhibit such a reaction, the individual must be able to employ his/her executive abilities to defuse the situation. The ability to defuse a hostile social situation requires a number of executive abilities including hypothesis generation, strategic planning, social problem-solving, and abstract reasoning, in order to develop a viable plan to deal with the situation in a non-aggressive manner. Once the plan has been formulated it must be implemented and the individual must remain resistant to

distracting or interfering stimuli through the use of attentional control. An inability to resist interference, for example, further provocations from the other individual, may cause a deviation from the intended plan, which may cause the individual to say or do something inconsistent with his or her desired goal (i.e., non-aggressive response). While the plan is being implemented, the individual must monitor internal and external states in order to determine how the plan is proceeding. Difficulties in external monitoring may interfere with the ability to read and correctly interpret potentially ambiguous social cues, which can lead to misunderstandings and misattributions and consequent inappropriate aggressive responses.

Finally, the individual must evaluate feedback information derived from self- and social-monitoring. If the feedback information is consistent with the goals of the plan (i.e., defusing the situation), the plan will continue to be executed until the goal is attained. If the feedback information indicates that the plan is not effective, the individual must be able to generate new plans and shift from one possible solution to the next until an adequate plan is devised and implemented. Low executive functioning in the form of cognitive inflexibility and poor hypothesis generation is likely to lead to rigid and concrete thinking. This will leave the individual unable to effectively deal with rapid changes in the environment that require novel, immediate solutions to resolve potentially aggressive situations. The failure to generate such solutions may prolong a hostile interaction, possibly leading to increased frustration and the propensity for heightened aggressive responses (Giancola, 2000).

5.10.9 Conclusion

Researchers have come to appreciate that the prefrontal region can be fractioned into anatomically and functionally distinct subsystems, namely the

orbitofrontal and dorsolateral systems. Following Lapierre et al. (1995), this study attempted to address this distinction by including neurocognitive tasks considered sensitive to dysfunction in the discrete dorsolateral subsystem, although, as noted previously, the localising value of such tasks remains controversial.

Overall, these results demonstrate that impulsive-aggressive individuals display impairments on tests of planning, problem-solving, flexibility of verbal thought processes and cognitive flexibility known to relate to dorsolateral prefrontal function. Consistent with previous literature, the predominantly premeditated-aggressive group did not differ from controls on measures of executive function. Thus, while previous studies have emphasised the role of the orbitofrontal cortex in impulsive-aggression, these results have also implicated the dorsolateral prefrontal cortex through its role in executive functions.

The overall finding that impulsive-aggressive individuals have impairments in executive functions fits with neuroimaging studies reporting selective structural (Raine et al., 2000) and functional prefrontal deficits (Goyer et al., 1994) in populations characterised by impulsive-aggression. The convergence of these findings suggests that the dorsolateral prefrontal cortex plays a key role in the expression of impulsive-aggression through the mediation of executive functions. An association between impulsive-aggressive behaviour and impairment in executive functions most probably reflects an inability to organise several parameters simultaneously and anticipate consequences of actions in order to solve interpersonal problems. The capacity to reflect on the interpersonal conflict may be overwhelmed when in social situations which call for a more adaptive response leading to the expression of impulsive-aggression (Block, 1995; Lapierre, et al., 1995).

The present results also indicate a pattern of personality differences between impulsive- and premeditated-aggressive individuals and non-aggressive controls. These results, in conjunction with previous work (Barratt et al., 1997b; Stanford et al., 2003b), suggest a distinctive personality style may be associated with aggressive behaviour, regardless of the type of aggression displayed. Considering these findings, it is hypothesised that the individual's capacity to control his/her behaviour plays a key role in the manifestation of aggressive behaviour. Those who display predominantly impulsive-aggressive behaviour are characterised by a number of cognitive deficits that seem to contribute to their loss of behavioural control. Conversely, premeditated-aggressive individuals appear to have an intact behavioural control system. These results thus provide some insight as to the underlying mechanisms of different aggressive subtypes, and allow for more accurate evaluation and treatment of such problem behaviour (Stanford et al., 2003a).

The finding of neuropsychological deficits in the current sample of impulsive-aggressive individuals is particularly striking in light of the general effectiveness and high functioning of this population. The impulsive-aggressive individuals in the current study, who, by societal standards, are functioning „normally', as opposed to incarcerated populations, have demonstrable neurocognitive weakness. Furthermore, their impulsive-aggressive behaviour is associated with specific neuropsychological deficits that are independent of brain trauma. While the sample size is relatively small, the consistency of the finding is suggestive of a shared pathology involving specific executive control processes. These findings are consistent with the neuropsychological and psychophysiological findings in impulsive-aggressive incarcerated criminals and support the notion of a specific behavioural syndrome associated with spontaneous aggressive outbursts (Barratt et al., 1997b).

In conclusion, the results of this study suggest that the dorsolateral prefrontal cortical dysfunction may contribute to the expression of impulsive-aggression. To further delineate the specific role of the prefrontal cortex in both impulsive- and premeditated-aggression, the following study will explore abilities localised to the orbitofrontal cortex, namely the ability to correctly interpret emotions in facial expressions and the attribution of emotions to ambiguous faces of emotion.

Chapter 6

Study 2: Emotion Recognition and Aggression Attribution

As previously discussed, a significant body of evidence indicates that the likelihood of acting aggressively is related to some functional capacity of the frontal lobe. Studies of violent offenders using both structural (Raine et al., 2000; Relkin, Plum, Mattis, Eidelberg & Tranel, 1996) and functional (Raine, et al, 1998; Søderstrom et al., 2000) imaging techniques have consistently shown abnormalities in frontal lobe structures in individuals who have histories of violence. Additionally, lesion studies (Damasio et al., 1994) and neuropsychological studies (Lapierre et al., 1995) have provided evidence of the relationship between the prefrontal cortex and the propensity for disinhibited aggressive behaviour.

To date, the predominant explanation for the demonstrated relationship between prefrontal abnormalities and aggressive behaviour has been executive dysfunction (Giancola, 2000). However, another possibility that has not been addressed in great detail is that aberrations in aspects of the prefrontal cortex may interfere with other cognitive and/or perceptual abilities. Disturbances of higher cognition and social behaviour have long been recognised as common sequelae of lesions or deficits in the prefrontal cortex. These behavioural changes have been linked with damage involving orbitofrontal or ventromedial prefrontal cortex, but the specific emotional, cognitive, and/or physiological processes that may be disrupted have not been well elucidated.

Several mechanisms have been proposed to explain the observed deficits in social behaviour following orbitofrontal or ventromedial damage lesions: impaired decision-making due to a lack of a „somatic marker' (Bechara et al., 1994; Damasio et

al., 1990); the inability to alter behaviour appropriately in response to a change in reinforcement contingencies (Rolls, 1996; Rolls et al., 1994); deficits in the ability to represent the mental states of others or „theory of mind' (Stone et al., 1998); or the ability to access social knowledge (Grafman et al., 1996; Hornak et al., 1996). Still other theorists have attributed orbitofrontal patients' poor social behaviour to an inability to use emotional information to guide behaviour (e.g., Bechara et al., 2000a).

Since the classic publication of Dodge (1986), social information processing has been assumed to constitute an important mechanism in the maintenance of aggressive behaviour patterns and therefore has often been formulated as the target of cognitive-behavioural interventions. If distinct forms of social information processing are found to characterise the different subtypes of aggression, this may have major implications for the interventions adopted in connection with aggressive behaviour.

One aspect of social behaviour needing further study is the ability to perceive and identify another person's affective or emotional state (affect perception). Facial expressions are non-verbal communicative displays vital in social cognition, allowing the transmission of information regarding internal emotional states and emotions to others in social situations (Blair et al., 1999). They can be considered as aspects both of an emotional response and of social communication (Adolphs, 2002a). In particular, one's ability to recognise facial expressions allows inferences to be made about moods and feelings, and in some cases may significantly influence the comprehension of language (Kolb, Wilson & Taylor, 1992).

The ability to adequately identify facial expressions of emotion is a problem that has been associated with a broad range of behavioural abnormality and psychopathology, including major depression (Surguladze et al., 2004), bipolar disorder (Getz, Shear & Strakowski, 2003; Venn et al., 2004), anorexia nervosa

(Kucharsak-Pietura, Nikolaou, Masiak & Treasure, 2004), schizophrenia (Kohler & Brennan, 2004), adult ADHD (Rapport, Friedman, Tzelepis & Van Voorhis, 2002), alcoholism (Frigerio, Burt, Montagne, Murray & Perrett, 2002; Townshend & Duka, 2003), and opiate dependence (Kornreich et al., 2003), all of which may or may not involve aggressive behaviour. Given this, an investigation into the specific relationship between aggression and emotion recognition is needed.

6.1 Neural systems involved in emotion recognition

Functional neuroimaging and lesions studies have identified neural systems responsive to human facial expressions (Davidson, Abercrombie, Nitschke & Putnam, 1999; Gur et al., 2002; Phillips et al., 1999; Wright, Martis, Shin, Fischer & Rauch, 2002). While it is recognised that there are areas in the primate temporal visual cortex where faces are represented, and there is a specialised population of neurons concerned with facial expression (Allison, Puse & McCarthy, 2000; Rolls, 1992), animal, neuroimaging, and human lesion studies consistently attribute impaired emotion recognition to dysfunction within a frontolimbic circuit that entails the orbitofrontal cortex, the amygdala, ventral striatum, and anterior cingulate (Phillips, Drevets, Rauch & Lane, 2003). There is a direct projection from the temporal visual cortex areas into the orbitofrontal/inferior frontal cortex. In addition, there is a route via the amygdala, where face-responsive cells are also found, to the orbitofrontal cortex (Rolls, 1990; 1999). Rolls, Critchley, Browning and Inoue (2006) also found a number of face-responsive neurons in the orbitofrontal cortex, and they are also present in adjacent prefrontal cortical areas. This suggests that information about faces, for example about expressions of emotion, may reach the orbitofrontal cortex,

where it could be used to provide reinforcing signals when social and emotional behaviour must be altered.

The suggestion of amygdala involvement is prompted by knowledge that this structure is crucial for processing threatening stimuli (e.g., Adolphs & Tranel, 2000; Adolphs et al., 1999; LeDoux, 1998). Calder et al. (1996) report on patient DR who had bilateral damage to the amygdala, and showed a recognition impairment for angry, fearful, disgusted, expressions. Similarly, Scott et al. (1997) report that the same patient was impaired in the recognition of the sounds of anger and fear, as well as angry, fearful, and sad tones of voice. Using fMRI with a facial matching and emotion identification task, Hariri, Bookheimer and Mazziotta (2000) reported that matching angry and frightened expressions was associated with bilaterally increased activation in the amygdala. However, cognitive processing of the facial expressions led to an attenuation of the amygdala response that was associated with engagement of the right prefrontal cortex.

Various frontal lobe regions have been active during a range of tasks involving emotional faces: the right anterior cingulate cortex and bilateral inferior frontal gyri during facial expression matching (George et al., 1993); the lateral orbitofrontal cortex and anterior cingulate cortex when matching fearful and neutral faces (Vuilleumrier, Armony, Driver & Dolan, 2001); the left ventral prefrontal cortex and left anterior cingulate cortex when making gender discriminations between angry faces (Blair et al., 1999); and the bilateral inferior frontal gyri during observation and imitation of emotional faces (Carr, Iacoboni, Dubeau, Mazziotta & Lenzi, 2003).

Nakamura et al. (1999) and Narumoto et al. (2000) reported right prefrontal response during elevation of facial expressions, while Sprengelmeyer et al. (1998) reported a study of six healthy adults performing a gender discrimination task of faces

expressing disgust, fear, and anger compared with neutral expressions. The fMRI data showed left inferior frontal activation for all three emotions, with different patterns for each emotion in other cortical and subcortical regions. More recently, Heberlein, Padon, Gillihan, Farah and Fellows (2008) compared emotion recognition performance of subjects with lesions confined to the ventromedial prefrontal regions to those with lesions elsewhere in the prefrontal cortex. They found that emotion recognition was impaired following ventromedial, but not dorsal or lateral, prefrontal damage. This impairment was demonstrated for the six emotions used in the task.

While some studies outlined above have linked impairments in recognising facial emotion to the prefrontal cortex broadly (Kolb & Taylor, 2000), most of the evidence points to those sectors of the prefrontal cortex connected with the amygdala and other structures that regulate emotion and autonomic function; principally ventral and medial sectors (Ongur & Price, 2000). Hornak et al. (1996) first explicitly demonstrated impaired recognition of emotion from facial expressions, and from the voice, following damage to the orbitofrontal cortex. Their patients had unilateral and bilateral damage to medial and lateral aspects of the orbital cortex (right unilateral damage was more frequently associated with impaired emotion recognition than was left unilateral damage) and such impairments occurred independently of perceptual impairments in facial recognition, voice discrimination or sound recognition. Poor performance on both expression tests was correlated with the degree of alteration of emotional experience reported by the patients. The authors also reported that patients' experience of certain emotions, particularly fear, decreased considerably following their lesion, suggesting that the orbitofrontal cortex may participate in both experience and recognition of emotion. A comparison group of patients with brain damage outside the ventral frontal lobe region, without these behavioural problems, was

unimpaired on the face expression identification tests, was significantly less impaired at vocal expression identification and reported little subjective emotional change.

The most detailed lesion study to date compared emotion recognition from face and vocal stimuli in groups with damage to the bilateral orbitofrontal cortex, unilateral orbitofrontal cortex, anterior cingulate cortex, and dorsolateral prefrontal cortex (Hornak et al., 2003). They found that some patients with bilateral lesions of the orbitofrontal cortex had deficits in voice and face expression identification, impairments in social behaviour and significant changes in their subjective emotional state. Patients with unilateral lesions of the antero-ventral part of the anterior cingulate cortex and/or medial prefrontal cortex were in some cases impaired on voice and face expression identification, had some change in social behaviour, and had significant changes in their subjective emotional state. Patients with dorsolateral prefrontal cortex lesions or with medial lesions outside the anterior cingulate cortex and medial prefrontal areas were unimpaired on any of these measures of emotion.

The results of Hornak et al. (2003) thus confirm that damage restricted to the orbitofrontal cortex can produce impairments in face and voice expression identification, both of which may be primary reinforcers. The system is sensitive, in that even patients with unilateral orbitofrontal cortex lesions may be impaired. The results also show that changes in social behaviour can be produced by damage restricted to the orbitofrontal cortex. The patients were particularly likely to be impaired on emotion recognition, emotional empathy (they were less likely to comfort those who were sad, or afraid, or to feel happy for others who are happy), interpersonal relationships (not caring what others think, and not being close to his/her family); and were less likely to cooperate with others; were impatient and impulsive, and had difficulty in making and keeping close relationships.

The significance of these specific neurons in the orbitofrontal cortex is likely to be related to the fact that faces convey information that is important in social interactons, both by conveying face expression, which can indicate reinforcement, and by encoding information about which individual is present (Rolls, 2007). This suggests that the orbitofrontal cortex is a vital system in emotion-related learning (Rolls, 1990; 1995).

Although it is clear that the ability to adequately identify facial expressions is not exclusively mediated by the prefrontal cortex, there is compelling neuroimaging evidence to suggest that this region plays a significant role in the successful identification of facial expressions of emotion. For example, Phan, Wager, Taylor and Liberzon (2002) conducted a meta-analysis of 55 PET and fMRI studies of neural regions activated by emotional stimuli (most typically facial expressions). They concluded that medial prefrontal cortex is most consistently activated by emotional stimuli, suggesting it has a central role in emotional processing.

6.2 Separable neural systems for different emotional expressions

Growing evidence suggests that the recognition of different emotional states involves at least partly separable neural circuits. Patients with bilateral damage to the amygdala show impaired processing of fear relative to other expressions of emotion (Adolphs, Tranel, Damasio & Damasio, 1994). Consistent with this, amygdala activation occurs specifically during the presentation of fearful facial expressions of emotion in healthy volunteers (Morris et al., 1996). Amygdala activation is also evident during perception of sad facial expressions (Blair et al., 1999). In contrast, the anterior insula and dorsal striatum are implicated in the processing of disgust (Gray, Young, Barker, Curtis & Gibson, 1997; Phillips et al., 1997, 1998).

Evidence concerning the processing of other key emotions, though limited, is growing. Neuroimaging implicates the medial frontal cortex in the processing of angry facial expressions (Blair et al., 1999; Phillips et al, 1999). Blair et al. (1999) found the right orbitofrontal cortex and anterior cingulate showed an enhanced response to angry expressions which correlated with expression intensity. The involvement of the anterior cingulate in the processing of anger accords with its well-known participation in the regulation of emotion and expression of emotional responses (Bush, Luu & Posner, 2000; Devinsky et al., 1995). Transcranial Magnetic Stimulation (TMS) research has supported this, finding that TMS over the medial frontal cortex impaired the processing of angry, but not happy, facial expressions of emotion. This effect was largest for difficult discriminations from neutral faces (Harmer, Thilo, Rothwell & Goodwin, 2001).

The medial and orbital regions are richly interconnected, especially via the ventromedial part of the frontal lobe, suggesting that a lesion in either might be expected to disrupt some of the same functions (Cavada, Company, Tejedor, Cruz-Rizzolo & Reinoso-Suarez, 2000; Koski & Paus, 2000; Ongur & Price, 2000). Nevertheless, these regions can be distinguished from each other by their specific pattern of connections with other parts of the brain, including sensory, limbic, and striatal-thalamic structures (Ongur & Price, 2000). To this extent, they would be expected to contribute differently to emotional processing.

The data thus indicate dissociable neural substrates that differentially responded to these distinct emotional expressions. These dissociable neural patterns are in keeping with the suggestion that there are distinct neural systems which respond to basic expressions of emotion (e.g., Adolphs, Damasio, Tranel & Damasio, 1996).

6.3 The relationship between emotion recognition and aggression

Theories of social competence postulate that social perception skills (i.e., facial affect recognition) are an important factor in mediating social skill and social adjustment (Wallace, 1984). Facial expressions of emotions are crucial in social cognition, communicating information to others, providing indicators of affective disposition in others and modulating behaviour according to the social context (Eimer & Holmes, 2007). In this way, activation of these neurons which are involved in facial expression recognition acts as a reinforcer. Certainly, in primate social interaction, individuals are constantly updating their evaluation of other individuals in terms of the reinforcers received. The rapid learning of associations between representations of expression and reinforcers in the orbitofrontal cortex is likely to be a vital part of this process.

There is a strong relationship between identifying emotional states and emotional experience. Adults have a tendency to mirror the facial expression of those whom they are interacting with (McHugo & Smith, 1996) and, in turn, lead to corresponding emotional changes (Levenson, Ekman & Friesen, 1990). Adults with focal frontal lesions and traumatic brain injury implicating the frontal lobes can experience reduced somatic responses to emotional material (Croker & McDonald, 2005; Hornak et al., 1996; McDonald, 2005; Saunders, McDonald & Richardson, 2006). Furthermore there is a relationship between responsivity and recognition accuracy (Croker & McDonald, 2005). This is consistent with Phillips et al. (2003) who argued that the ventral frontal system mediates both the early appraisal of emotionally significant stimuli, as well as the affective responses to those stimuli.

The display of sad expressions has long been linked to the inhibition of aggression and the elicitation of prosocial behaviour (e.g., Miller & Eisenberg, 1988;

Eisenberg et al., 1989). Following with this, Blair (1995) postulated the existence of a violence inhibition mechanism in humans which would be activated by non-verbal communication of distress, including sad or fearful facial expressions. Angry facial expressions, on the other hand, are displayed to curtail the behaviour of others in situations where they have broken the social rules or social expectations (Averill, 1982). They are also thought to be important signals to modulate current behavioural responding, particularly in situations involving hierarchy interactions (Blair & Cipolotti, 2000; Lerner & Keltner, 2001).

As accurate interpretation of facial expressions is important for social interaction, one would expect that individuals who have trouble interpreting facial expressions of emotions would be less socially competent and fail to adequately modulate behaviour according to social context (Weiss et al., 2006). Misinterpretations of social cues, such as angry and fearful facial expressions, may lead to inappropriate reactions in social interactions, such as acting in a way that causes harm to others. In addition, such individuals might be unaware that their behaviour unintentionally engenders fear in others. Aggressive behaviour may thus be a result of a failure to benefit from restraint-producing environmental cues that serve a regulatory role, such as facial signs of anger.

In a variety of studies, it has been shown that distortion or misinterpretation of social cues can result in the generation of inappropriate social responses, such as reacting aggressively or violently in ambiguous social situations (Akhtar & Bradley, 1991; Dodge et al., 2002). The most comprehensive discussion of social information processing and its relationship with aggressive behaviour comes from Dodge (1986), and a reformulation of this model by Crick and Dodge (1994). The models propose that to react appropriately to social situations, social information has to be processed

in an orderly fashion: (1) the information has to be coded accurately; (2) the encoded information has to be represented correctly; (3) an interaction goal needs to be specified; (4) response alternatives have to be generated; (5) response alternatives have to be evaluated and, from these responses, an optimal response has to be selected; and (6) the selected response has to be enacted. These initial stages of the model thus constitute low-level processing and encoding of the affective cues of others.

According to Dodge (1980), one of the reasons why aggressive children act aggressively is that they attribute hostile intentions to others more often than do other children. This phenomenon has become widely known as „hostile attribution bias'. Aggressive children have, in fact, been shown repeatedly to interpret ambiguous social cues as hostile (Crick & Dodge, 1996; Dodge & Schwartz, 1997; Orobio de Castro, Veerman, Koops, Bosch & Monshouwer, 2002) which suggests that deviant social information processing plays a role in the development and maintenance of aggressive behaviour.

Different aspects of social information processing may play a role in the manifestation of impulsive- versus premeditated-aggression (Dodge, 1991). According to the frustration-aggression theory typically applied to impulsive-aggression, this form of aggression arises predominantly among individuals who show a strong orientation towards negative information during the initial stages of information processing, a tendency to interpret this information as threatening and frustrating, and a propensity to become emotionally aroused as a result of their interpretation of the social information. Via a combination of insufficient information processing, temperament, threatening experiences and difficulties in emotion

regulation, impulsive-aggressive individuals experience a greater number of situations as frustrating and in turn react aggressively.

In contrast, according to the social-learning theory typically applied to premeditated-aggression, distortions arise during the later stages of social information processing as previous learning experiences have made aggressive behaviours quickly and easily accessible and aggression is expected to produce positive outcomes. That is, premeditated-aggression is displayed as a result of positive experiences with aggression in other situations (Merk et al., 2005).

Facial expressions are one way to assess emotion-relevant attributional bias, and researchers have demonstrated that individuals perceive pictures of facial expressions in a way that is consistent with their perceptual set (Maner et al., 2005). Hall (2006) found a significant positive correlation between errors of commission in regard to identification of anger from photographs and aggressive attitude and verbal aggression in a sample of undergraduate students. She argued that such individuals appeared to be primed to perceive the world based on their own negative schema and to interpret information around them to conform to these biases. Such findings are consistent with those of previous studies indicating that people often interpret the behaviour of others based on their own internal feelings and beliefs (Dodge et al., 2003; Hubbard et al., 2001).

Regarding these self-schemas, it has been suggested that individuals link past experiences to form dominant themes, and that these guide cognitive processing and social interactions (Milne & Grafman, 2001). According to Blackburn (1989), with widespread experiences, these emotional scripts become over-generalised into social interactions. In the case of aggressive individuals, this bias is an attribution of hostile intent in social interactions. Novaco and Welsh (1989) propose a similar concept,

known as the social-information processing theory, describing the relationship between information processing deficits and aggression. These pre-existing belief systems, or schemas, influence what a person attends to and how they encode and store the information. For example, aggressive reactions to an aversive interpersonal situation can increase an individual's expectancy that others will behave aversively. These expectancies in turn can effectively increase the likelihood that the individual will react in an aggressive manner in future interpersonal situations that are not necessarily of an aversive nature (Huesmann, 1988). Dodge and Tomlin (1987) provide support for this theory finding that when participants were asked to explain their decision as to the intent they attributed to peer provocation, aggressive participants were more likely to utilise self-schemas rather than the provided cues.

Social cognitive models propose that this cognitive mediation is a relatively automatic process as these individuals will act in accordance with their own schemas, which have been learnt and rehearsed throughout life. In support of this hypothesis, Dodge and Newman (1981) found that when aggressive subjects were encouraged to respond faster, they made greater attributions of hostile intent in interpreting frustrating situations when the reason for the conflict was ambiguous. In addition to this, Dodge and Coie (1987) found that only impulsive-aggressive children and not instrumental-aggressive children demonstrated hostile biases in their attributions of peer intentions in provocative situations. It thus appears that faster latency of responding facilitates self-schemas of hostile attributions in aggressive subjects.

Deficiencies in processing and responding to social cues increase the likelihood of emergent maladaptive or inappropriate behaviours, as cognitive appraisals of events influence behavioural reactions displayed by the individual. These reactions then modify subsequent cognitive appraisals of events (Novaco &

Welsh, 1989). This self-perpetuating cycle is demonstrated in aggressive reactions to an aversive interpersonal situation increasing the individual's expectation that others will behave aversively. Such expectancies in turn effectively lower the threshold of additional aggressive reactions. Cognitive factors and processes may either amplify or attenuate aggressive reactions to aversive or negative events. Often, however, excessive and inappropriate aggression disrupts non-aversive interpersonal situations.

Insight into the relationship between cognitive processes and behavioural reactions may provide important insights into the potentially significant cognitive characteristics of individuals who repeatedly react aggressively in interpersonal situations. This bias not only encompasses their appraisals of aversive interpersonal situations, but also distorts their appraisal of benign situations. The latter effect may prove to be particularly problematic, since a bias to attribute hostility might easily increase incorrect appraisals of both aversive interpersonal situations without hostile intent and non-aversive interpersonal situations, which do not warrant such attributions (Nasby, Hayden & DePaulo, 1980).

As discussed previously, the orbitofrontal cortex has been implicated in the recognition of emotional expressions (Hornak et al., 1996). The orbitofrontal cortex is also shown to be involved in the mediation of behavioural extinction and the reversal of behaviour to stimuli (e.g., faces) when the reinforcement contingencies change (Dias et al., 1996a; Rolls, 1996). It thus has an important role in social and emotional behaviour. Blair et al. (1999) suggest that the orbitofrontal cortex response to angry expression observed in their study is a reflection of a behavioural extinction/response reversal effect of these stimuli. They propose that the orbitofrontal cortex, when activated by angry expression stimuli, acts to suppress current behaviour through inhibition or by activation of alternative behavioural responses. In this way, angry

expressions are displayed to inhibit the behaviour of others in social situations and, in this sense, effectively terminate the ongoing behaviour of others (Averill, 1982). Angry expressions thus serve as a cue for behaviour extinction and/or reversal learning.

In normal social and emotional behaviour, humans must continuously react to the signals provided by facial expressions and emotions are often considered to be states elicited by rewarding and punishing stimuli. For example, the fear is a state elicited by stimuli learned to be associated with punishment, and joy is a state associated with rewarding stimuli. Because emotions are related to rewarding and punishing events which ultimately alter the probability of behaviour occurring, any failure to correct behaviour when the reinforcing value of environmental stimuli changes will lead to inappropriate emotional and social behaviour. A tendency to attribute hostility to ambiguous stimuli within the environment may contribute to aggressive behaviour. As impulsive-aggressive individuals may be more likely to encounter aggressive social cues through their misattribution of ambiguous cues, these individuals are consequently likely to respond in a similarly aggressive manner to both aggressive and neutral cues, compared with other individuals. Furthermore, this response could be a „first-strike' self-defensive action common to individuals with impulsive-aggressive personality traits.

Research supports the association between face decoding deficits and subjective emotional changes and observable changes in behaviour. Hornak et al. (1996) found that the greater total alteration in emotional experience (increase or decrease in both positive and negative emotions) as reflected in a subjective emotional changes questionnaire, the worse the performance on each of the tests of expression

identification. This correlation suggests that for some patients with frontal damage, the expression identification deficit may be part of a wider emotional disturbance.

Individuals with disorders marked by antisocial behaviour frequently show impairment in recognising displays of facial affect. For example, Blair and Cipolotti (2000) reported on a patient with sociopathy and episodic aggression following a right hemisphere trauma who experienced severe difficulty in the recognition of angry and disgusted facial expressions. Walz and Benson (1996) demonstrated that aggressive men with mental retardation did not have greater difficulty with emotion labelling and discrimination compared to peers, but had a negative emotional bias for ambiguous facial expressions. Aggressive subjects were more likely to state that a face was expressing anger when they were unsure of the emotion.

These findings are consistent with Dodge's (1993) and McNiel, Eisner and Binder's (2003) model of social information processing, in which they predict that a cognitive style characterised by hostile attributions increases the risk of violence. Although Dodge and McNeil et al. did not study facial expressions of emotion directly, they found that in ambiguous situations, aggressive individuals attend to fewer emotional cues, interpret actions and intentions of others as involving anger, and display deficits in what is described as affective perspective taking.

More recently, Mah, Arnold and Grafman (2005) compared the performance of patients with ventromedial prefrontal cortex or dorsolateral prefrontal cortex lesions with healthy volunteers on the Tests of Social Intelligence (O'Sullivan & Guilford, 1976). As hypothesised, only patients with ventromedial prefrontal lesions showed specific impairments in the ability to use non-verbal cues to interpret emotional expression, as well as the ability to complete a socially meaningful story by interpreting the feelings and behaviour of characters. The authors argued that the

results suggested deficient social knowledge, namely difficulty interpreting non-verbal emotional expression, contribute to the aberrant social behaviour observed following ventromedial prefrontal cortex lesions.

Mitchell, Avny and Blair (2006) presented results from patient CL, who had „acquired psychopathy' following an orbitofrontal cortex lesion. CL showed impairment in the identification of fearful, happy, surprised, disgusted, and angry facial expressions, and in comparison to psychopathic individuals, CL was significantly impaired for happy and angry facial expressions. In combination with their other findings, the authors concluded that a failure to recognise disapproving social cues will compromise the ability to adjust behaviour in response to changing social demands.

While the above studies have demonstrated a relationship between emotion recognition deficits and antisocial behaviour in individuals with damage to the orbitofrontal or ventromedial prefrontal cortex, only one study has investigated the relationship in aggressive individuals without neurological damage specifically. Hoaken et al. (2007) investigated possible facial expression identification deficits in violent and non-violent offenders. They found that the violent offender group was significantly poorer at the interpretation of facial expressions of emotion than either non-violent offenders or controls. When they assessed which attributions the groups made of neutral faces, they found that the non-violent and control groups were most likely to attribute an emotion seemingly irrelevant to aggression (most often „sadness'), whereas violent offenders were more likely to respond „disgust'. Although this response does not clearly demonstrate a tendency of these participants to systematically interpret hostility, it does suggest that they have a tendency to less

positively interpret the emotions of others. This can be seen as theoretically consistent with a propensity for acting aggressively.

In sum, the preceding evidence suggests that aggressive reactions are often prompted by an interpretation that another person is acting with hostile intent towards the individual. Characteristic impulsivity may strengthen the tendency to make such attributions through faster responding. Impulsive-aggression can also be characterised as a defensive reaction to a perceived threatening stimulus. The perception of threat and experience of anger consequently causes the individual to act aggressively, either in defence or in retaliation. Errors and biases in interpreting threats are thus hypothesised to account for the inappropriate display of impulsive-aggression.

6.4 Aim and hypotheses

The present study investigated emotion recognition in impulsive-aggressive and premeditated-aggressive individuals in comparison to controls using an emotion recognition task and an aggression rating task. Given the scarcity of research on emotion recognition in aggressive populations, the current study sought to investigate whether impulsive-aggressive individuals show greater impairment in one aspect of social cognition, namely the ability to interpret facial expressions of emotion, relative to premeditated-aggressive individuals and controls. As the predominant focus of the current research is on the role of the orbitofrontal cortex, a particular focus was on the interpretation of aggressive and neutral faces. Possible dysfunction in the interpretation of other emotions would also be explored.

A relationship between orbitofrontal damage and the display of impulsive-aggression has been established (e.g., Rolls et al., 1994). Furthermore, individuals with damage to the orbitofrontal cortex have been shown to misinterpret facial

expression of emotion (e.g., Hornak et al., 1996). It was therefore hypothesised that impulsive-aggressive individuals, due to possible dysfunction in orbitofrontal functioning, would be impaired on facial expression recognition, specifically the interpretation of aggressive and possibly neutral expressions, relative to premeditated-aggressive individuals and controls.

Furthermore, impulsive-aggressive individuals have been shown to misattribute hostile intent in benign social situations. Thus, on the aggression rating task, it was hypothesised that the impulsive-aggressive individuals would rate the neutral faces as significantly more aggressive than would the premeditated-aggressive and control participants.

Differences in responses at the 1000ms and 2000ms stimulus durations on the emotion recognition task and aggression rating task would be investigated to determine if shorter processing time hinders the correct interpretation of the faces. This is based on Dodge and Coie's (1996) argument that faster latency of responding would facilitate a reliance on self-schemas and therefore a hostile attribution of intent.

6.5 Method

6.5.1 Participants

An initial pool of 435 students from the University of Tasmania were screened using the BPAQ-SF (Bryant & Smith, 2001). The sample was recruited from a different cohort than that in Study 1. Aggression scores were non-normally distributed, with skewness of .534 (*SE* = .118) and kurtosis of -.583 (*SE* = .23).

Based on questionnaire responses, 100 participants (female = 59, male = 41) were selected and placed into one of two groups; aggressive (*n* = 70), and non-aggressive controls (*n* = 30). Participants were selected and grouped according to the

selection criteria detailed in Chapter 5. The aggression scores differed significantly between the aggressive group (M = 37.3, SD = 4.82) and control group (M = 15.27, SD = 1.93), F (1, 87) = 584.01, MSE = 10208.05, p<.001, η^2 = .856. Using the IPAS (Stanford et al, 2003a), the aggressive group was further divided into predominantly impulsive-aggressive and predominantly premeditated-aggressive utilising the grouping criteria as for Study 1. This method led to the exclusion of 13 participants from the 70 aggressive individuals initially identified. Further exclusion criteria for this study are outlined in Chapter 5. The final sample consisted of 35 impulsive-aggressive, 22 premeditated-aggressive and 30 control participants. The number of males and females in each group is shown in Table 6.1.

Table 6.1

Number of males and females in the three participant groups and total sample

	Impulsive-Aggressive	Premeditated-Aggressive	Control	Total
Males	13	13	11	37
Females	22	9	19	50

Mean aggression scores on the BPAQ-SF for the three participant groups are shown in Table 6.2. There was a significant difference in aggression scores between the three groups, F(2, 86) = 299.26, MSE = 4637.95, p<.001, η^2 = .877. Post hoc Tukeys indicated that the impulsive-aggressive and premeditated-aggressive groups had significantly higher aggression scores than the control group (ps<.05). There was no significant difference in BPAQ-SF scores between the males and females in the impulsive-aggressive group, F(1, 34) = .004, MSE = .12, p = .95, η^2 = .000,

premeditated-aggressive group, $F(1,21) = 1.01$, MSE = 14.03, $p = .33$, $\eta^2 = .048$, control group, $F(1, 29) = 1.99$, MSE = 7.17, $p = .17$, $\eta^2 = .066$, or total sample, $F(1, 86) = .92$, MSE = 113.55, $p = .34$, $\eta^2 = .011$.

Participants' ages ranged from 17 to 30 years with a mean age of 19.86 years (*SD* = 2.72). Mean ages for each participant group are presented in Table 6.2. The premeditated-aggressive group was significantly older than the impulsive-aggressive and control groups, $F(2, 87) = 4.02$, MSE = 27.72, $p = .02$, $\eta^2 = .087$.

Table 6.2.

Mean (and standard deviations) scores on the Aggression Questionnaire – Short Form and ages for the three participant groups and total sample

	Impulsive-Aggressive	Premeditated-Aggressive	Control	Total
BPAQ-Revised	37.46 (5.15)	36.18 (3.72)	15.27 (1.93)	29.48 (11.09)
Males	37.50 (4.66)	35.22 (3.42)	14.89 (1.97)	30.81 (10.72)
Females	37.38 (6.10)	36.85 (3.91)	15.91 (1.76)	28.50 (11.36)
Age	19.49 (1.92)	21.23 (3.94)	19.30 (2.10)	19.86 (2.72)

6.5.2 Materials

6.5.2.1 Questionnaires

The BPAQ-SF (Bryant & Smith, 2001) was used for the purpose of selecting participants who were characteristically aggressive. The IPAS (Stanford et al., 2003a) was employed to characterise the aggressive acts as predominantly impulsive aggressive or predominantly premeditated in nature. Participants also completed the BPAQ (Buss & Perry, 1992) and I7 Impulsivity Questionnaire (Eysenck et al., 1985)

upon completion of the computer tasks. All questionnaires are outlined in detail in Chapter 5.

6.5.2.2 Facial recognition tasks

Emotion recognition task: Stimuli for the emotion recognition task comprised 56 faces conveying one of seven different emotions: sad, angry, disgusted, surprised, frightened, happy and neutral. Faces were black and white photographs obtained from Ekman and Friesen's (1976) slides, which were digitised for computer presentation. Faces were selected from the Ekman and Friesen pool based on piloting studies in which individuals (n = 100) were asked to indicate which facial expression was being depicted by choosing from a list of seven expressions (aggressive, disgusted, frightened, happy, neutral, surprised, sad). Faces identified with most accuracy were chosen for the tasks. The final 56 face stimuli was made up of four female faces and four male faces, each portraying one of each emotion listed above (see Appendix D for facial stimuli). The Ekman and Friesen faces have been widely used and validated as accurate depictions of emotional affect, and thus provide a reliable and valid means of assessing facial-affect recognition deficits.

Aggression rating task: Following the emotion recognition task, participants completed an aggression-rating task. The task consisted of 50 facial stimuli obtained from Ekman and Friesen's (1976) collection, depicting one of five facial expressions: aggressive, disgusted, frightened, happy, or neutral. The five female faces and five male faces each portrayed one of each emotion listed above. The facial stimuli chosen for this task were taken from the pilot study described above (see Appendix E for facial stimuli).

Both tasks were presented on a Pentium 90 computer with a 15" monitor. Participants used the computer keyboard to indicate their responses.

6.5.2.3 Wechsler Adult Intelligence Scale – Third Edition (Wechsler, 1997)

Two subtests from the WAIS-III were selected as control measures. As outlined in Chapter 5, Vocabulary was used as a measure of general intellectual functioning, while Digit Span was used as a measure of verbal working memory and attentional capacity for verbal information (Wechsler, 1997).

6.5.3 Procedure

The BPAQ-SF was used to recruit participants from undergraduate psychology classes at the University of Tasmania. Following the screening process, participants who qualified as aggressive or control participants were invited to participate in the emotion recognition and aggression rating tasks, as well as the tasks completed for Study 3 (outlined in Chapter 7). Approval from the Human Research Ethics Committee (Tasmania) Network was obtained before the recruitment procedures took place (see Appendix A for approval letter).

All participants received course credit for their participation. Informed written consent was obtained from all individuals prior to participation (see Appendix F and G for participant information sheets and consent forms). Participants were tested individually in a quiet room in the School of Psychology at the University of Tasmania. Participants completed the tasks for Study 2 and Study 3 in the same testing session. Presentation of the tasks was counterbalanced.

Participants were seated in front of a computer monitor, approximately 60cm from the screen. The emotion recognition task was explained and participants were instructed to respond to the face by pressing the corresponding key to the emotion that

they thought the face was depicting. Participants used both of their index, middle, and ring fingers on the numbers one through to six on the number keys at the top of the keyboard to indicate their response.

The emotion recognition task entailed a face being presented centrally for either 1000ms or 2000ms, followed by a list of six emotions (sad, angry, frightened, disgusted, surprised, happy), numbered one through to six. The order in which the emotion list was presented was randomised for each face, and the stimulus duration was counterbalanced. The faces were approximately 220 mm in height and 140 mm wide, presented on a white background. The inter-trial interval was 1500ms. The task consisted of 56 trials. The dependent variables for this task were the number of incorrect responses to the non-neutral stimuli, and the nature of responses to the neutral stimuli.

Participants then completed the aggression rating task. This task entailed a face being presented centrally on the computer screen for either 1000ms or 2000ms (counterbalanced order), followed by a 5-point Likert scale (1 = not at all aggressive, 2 = a little aggressive, 3 = quite aggressive, 4 = very aggressive, 5 = extremely aggressive). Participants placed their two index fingers, two middle fingers, and left ring finger on the numbers one through to five at the top of the keyboard and were required to press the corresponding number to the perceived level of aggressiveness for each face. The inter-trial interval was 1500ms. The task consisted of 50 trials. The dependent variable was mean aggressiveness ratings of each face type.

Following completion of the computer tasks for Study 2 and Study 3, participants completed the WAIS-III Vocabulary and Digit Span subtests, and the BPAQ, IPAS, and I7 Impulsivity questionnaires. Participants were fully debriefed at the end of the testing session.

6.6 Results

6.6.1 Participants

Mean scores on the BPAQ and I7 Impulsivity Questionnaire were analysed using separate one-way ANOVAs. On the BPAQ, a significant effect was found for the physical aggression, $F(2, 86) = 94.44$, $MSE = 1841.3$, $p<.001$, $\eta^2 = .692$, verbal aggression, $F(2, 86) = 82.98$, $MSE = 830.93$, $p<.001$, $\eta^2 = .664$, hostility, $F(2, 86) = 44.32$, $MSE = 1058.84$, $p<.001$, $\eta^2 = .513$, anger subscales, $F(2, 86) = 178.23$, $MSE = 1514.63$, $p<.001$, $\eta^2 = .809$, and total aggression, $F(2, 86) = 343.15$, $MSE = 20187.46$, $p<.001$, $\eta^2 = .891$. Post hoc Tukeys indicated that the impulsive-aggressive and premeditated-aggressive groups had significantly higher scores than the control group on all subscales (*p*s<.05). Comparable to the mean aggression scores for the sample in Study 1, the mean scores of the impulsive-aggressive and premeditated-aggressive are analogous to Smith and Waterman's (2004) sample of violent offenders.

On the I7 Impulsivity Questionnaire, a significant effect was found for the combined impulsivity-venturesomeness subscale, $F(2, 86) = 3.14$, $MSE = 161.74$ $p = .048$, $\eta^2 = .070$, impulsivity, $F(2, 86) = 7.77$, $MSE = 149.38$ $p = .001$, $\eta^2 = .156$, and empathy, $F(2, 86) = 6.04$, $MSE = 62.99$, $p = .004$, $\eta^2 = .126$. No significant effect was found for venturesomeness, $F(2, 86) = .1$, $MSE = 1.83$, $p = .9$, $\eta^2 = .002$. Post hoc Tukeys indicated that on the combined subscale, there was no significant difference between the groups (*p*s>.05), however a trend was shown for the impulsive-aggressive group to have significantly higher scores than the control group. On the impulsivity subscale, the impulsive-aggressive group had significantly higher scores than the premeditated-aggressive and control groups (*p*s<.05). On the empathy subscale, the premeditated-aggressive group had significantly lower scores than the

impulsive-aggressive and control groups (*p*s<.05). Mean scores for both questionnaires are shown in Table 6.3.

Table 6.3

Means (and standard deviations) for the subscales of the Aggression Questionnaire – Full Scale and I7 Impulsivity Questionnaire for the three participant groups

Subscale	Group			Total
	Impulsive-Aggressive	Premeditated-Aggressive	Control	
BPAQ				
Physical	23.91 (5.03)	26.31 (5.55)	11.3 (2.12)	20.17 (7.87)
Verbal	17.62 (3.81)	19.00 (3.41)	9.03 (1.88)	15.01 (5.39)
Hostility	24.20 (5.84)	22.27 (4.84)	13.20 (3.51)	19.92 (6.93)
Anger	22.20 (2.96)	21.31 (3.93)	9.47 (1.78)	17.59 (6.60)
Total	87.94 (9.11)	88.91 (7.46)	43.00 (5.74)	72.69 (22.96)
I7				
Imp-Vent	20.31 (6.72)	17.77 (7.06)	15.87 (7.74)	18.14 (7.35)
Impulsivity	10.97 (3.97)	8.14 (4.02)	6.77 (5.06)	8.80 (4.72)
Venturesomeness	9.34 (4.01)	9.64 (4.56)	9.10 (4.29)	9.33 (4.21)
Empathy	15.00 (3.20)	12.05 (4.10)	14.50 (2.46)	14.08 (3.41)

6.6.2 Emotion recognition task

Response: Mean number of correct responses were analysed with three-way repeated measures ANOVAs for group by face type by stimulus duration (see Table 6.4 for means and standard deviations).

Table 6.4

Mean (and standard deviations) number of correct responses (maximum = 4) on the emotion recognition task for the three participant groups

Face Type	Group			Total
	Impulsive Aggressive	Premeditated Aggressive	Control	
Aggressive				
1000ms	2.86 (0.94)	2.91 (0.75)	2.97 (0.67)	2.91 (0.80)
2000ms	3.00 (0.73)	3.09 (0.75)	3.10 (0.75)	3.06 (0.74)
Total	2.92 (0.56)	3.00 (0.56)	3.03 (0.45)	2.98 (0.52)
Disgusted				
1000ms	2.94 (1.19)	2.95 (1.09)	2.97 (0.85)	2.95 (1.04)
2000ms	2.63 (1.09)	2.41 (1.05)	2.67 (1.18)	2.59 (1.11)
Total	2.79 (0.83)	2.68 (0.91)	2.82 (0.74)	2.77 (0.81)
Frightened				
1000ms	2.63 (1.21)	2.45 (1.37)	2.40 (1.22)	2.51 (1.25)
2000ms	2.86 (1.09)	2.50 (1.14)	2.73 (1.17)	2.72 (1.13)
Total	2.74 (0.97)	2.48 (1.18)	2.57 (1.02)	2.62 (1.04)
Happy				
1000ms	3.91 (0.28)	3.81 (0.39)	3.80 (0.41)	3.85 (0.36)
2000ms	3.39 (0.32)	3.77 (0.43)	3.93 (0.25)	3.87 (0.33)
Total	3.90 (0.20)	3.80 (0.30)	3.87 (0.22)	3.86 (0.24)
Surprised				
1000ms	3.37 (0.65)	3.36 (0.73)	3.47 (0.73)	3.40 (0.69)
2000ms	3.03 (1.10)	3.36 (1.00)	3.53 (0.57)	3.29 (0.94)
Total	3.20 (0.77)	3.36 (0.77)	3.50 (0.57)	3.35 (0.71)
Sad				
1000ms	2.97 (1.10)	2.95 (1.13)	3.03 (1.10)	2.99 (1.09)
2000ms	3.00 (0.77)	3.05 (0.95)	3.97 (0.81)	3.00 (0.82)
Total	2.99 (0.66)	3.00 (0.89)	3.00 (0.87)	2.99 (0.79)

Analyses for correct response revealed a significant effect of face type, F(4, 84) = 32.46, MSE = 47.44, $p<.001$, $\eta^2 = .279$. Paired-samples *t*-tests indicated that participants made significantly more correct responses when interpreting the happy face than the five other face types. Surprised faces were interpreted with significantly more accuracy than the aggressive, disgusted, frightened and sad faces, the sad faces were interpreted with significantly more accuracy than the frightened face, and the aggressive face was interpreted with significantly more accuracy than the frightened and disgusted faces (all *p*s<.05). While some of these results would not remain significant after bonferroni adjustment, this adjustment can be regarded as too conservative, and thus will not be adopted in the current analyses (Howell, 2007). See Table 6.5 for results of *t*-tests.

A significant face type by stimulus duration interaction was also found, F(4, 84) = 3.30, MSE = 2.26, $p = .011$, $\eta^2 = 038$ (see Figure 6.1). Paired samples *t*-tests indicated that participants were significantly more accurate when interpreting the disgusted face at the 2000ms stimulus duration than the 1000ms stimulus duration, $t(86) = 2.42$, $p = .017$. There were no significant differences between responses at the 1000ms and 2000ms stimulus durations for the aggressive, $t(86) = -1.23$, $p = .223$, frightened, $t(86) = -1.76$, $p = .082$, happy, $t(86) = -.42$, $p = .672$, surprised, $t(86) = 1.3$, $p = .198$, or sad faces, $t(86) = -.10$, $p = .924$. No other significant main effects or interactions were found for response on the emotion recognition task.

In order to assess the nature of the responses to the eight neutral faces, a MANOVA was conducted to investigate whether the groups differed on frequencies of the six emotions participants attributed to the neutral faces. That is, a count was conducted for each of the participant's responses to the neutral faces as being „angry', „happy', etc (see Table 6.6 for mean frequency of ratings for each face type).

Table 6.5

Paired samples t-test results for response for each face type

Face 1	Face 2	*t*	df	*p*
Aggressive	Disgusted	2.02	86	.047*
	Frightened	3.06	86	.003**
	Happy	-14.25	86	.000***
	Surprised	-3.86	86	.000***
	Sad	-.118	86	.907
Disgusted	Frightened	1.18	86	.241
	Happy	-12.88	86	.000***
	Surprised	-4.90	86	.000***
	Sad	-1.85	86	.068
Frightened	Happy	-11.13	86	.000***
	Surprised	-4.79	86	.000***
	Sad	-3.04	86	.003**
Happy	Surprised	6.55	86	.000***
	Sad	9.97	86	.000***
Surprised	Sad	2.88	86	.005**

* $p<.05$, ** $p<.01$, *** $p<.001$.

At the separate 1000ms and 2000ms stimulus durations, no significant group differences were found (all *p*s>.05) (see Table 6.7 for results of ANOVAs). When the stimulus durations were collapsed, the groups differed in how frequently they interpreted the neutral faces as displaying frightened, $F(2, 84) = 3.23$, MSE = .59, $p = .045$, $\eta^2 = .071$. Post hoc Tukeys indicated that the premeditated-aggressive group were more likely to interpret the neutral faces as frightened than controls (*p*s<.05). No group differences were found for the aggressive, $F(2, 84) = 1.7$, MSE = 4.46, $p = .188$, $\eta^2 = .039$, disgusted, $(2, 84) = 1.36$, MSE = 1.37, $p = .262$, $\eta^2 = .031$, happy, (2,

84) = .103, MSE = .40, p = .902, η^2 = .002, surprised, (2, 84) = 1.07, MSE = .28, p = .347, η^2 = .025, or sad, (2, 84) = 1.78, MSE = 7.42, p = .175, η^2 = .041, facial expressions.

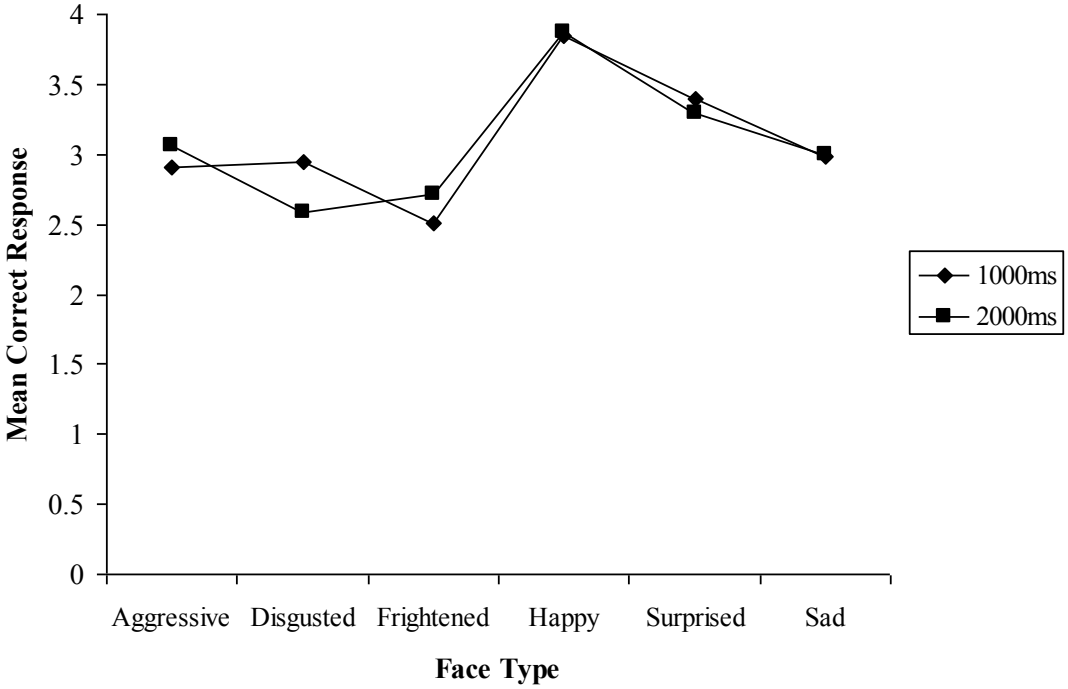

Figure 6.1. Mean number of correct responses (maximum = 4) for each face type at the 1000ms and 2000ms stimulus durations.

To test the hypothesis for this task, paired-samples *t*-tests were conducted to determine the face type participants attributed to the neutral face most often. As shown in Table 6.8, participants attributed „sad' to the neutral faces most often and significantly more than the other five face types.

Table 6.6

Mean (and standard deviations) frequency of responses to the neutral face on the emotion recognition task for the three participant groups

Face Type	Group			Total
	Impulsive Aggressive	Premeditated Aggressive	Control	
Aggressive				
1000ms	0.83 (0.99)	1.00 (1.02)	0.77 (0.99)	0.85 (0.96)
2000ms	0.80 (0.93)	1.27 (1.16)	0.70 (0.84)	0.89 (0.98)
Total	1.63 (1.61)	2.27 (1.83)	1.47 (1.46)	1.74 (1.63)
Disgusted				
1000ms	0.31 (0.58)	0.05 (0.80)	0.47 (0.73)	0.41 (0.69)
2000ms	0.29 (0.46)	0.46 (0.67)	0.50 (0.78)	0.40 (0.64)
Total	0.60 (.78)	0.96 (1.05)	0.97 (1.19)	0.82 (1.01)
Frightened				
1000ms	0.03 (0.17)	0.14 (0.35)	0.03 (0.18)	0.06 (0.23)
2000ms	0.11 (0.32)	0.23 (0.43)	0.03 (0.18)	0.12 (0.32)
Total	0.14 (0.36)	0.36 (0.66)	0.07 (0.25)	0.17 (0.44)
Happy				
1000ms	0.91 (1.15)	0.64 (0.85)	0.70 (1.06)	0.77 (1.04)
2000ms	.091 (1.12)	0.96 (1.29)	1.00 (1.11)	0.95 (1.15)
Total	1.83 (2.08)	1.59 (1.74)	1.70 (1.95)	1.72 (1.93)
Surprised				
1000ms	0.17 (0.45)	0.14 (0.35)	0.10 (0.40)	0.14 (0.41)
2000ms	1.14 (0.36)	0.05 (0.21)	0.03 (0.18)	0.08 (0.27)
Total	0.31 (0.58)	0.18 (0.50)	0.13 (0.43)	0.22 (0.52)
Sad				
1000ms	1.74 (1.40)	1.59 (1.10)	1.93 (1.23)	1.77 (1.26)
2000ms	1.74 (1.17)	1.05 (1.13)	1.73 (1.26)	1.56 (1.22)
Total	3.49 (2.29)	2.64 (1.71)	3.67 (1.95)	3.33 (2.06)

Table 6.7

ANOVA results for responses to the neutral face for the main effect of participant group at the 1000ms and 2000ms stimulus durations

Face Type	df	F	MSE	*p*	η^2
Aggressive					
1000ms	2, 84	0.39	0.36	0.68	.009
2000ms	2, 84	2.46	2.29	0.09	.055
Disgusted					
1000ms	2, 84	0.62	0.3	0.54	.014
2000ms	2, 84	1.01	0.41	0.37	.024
Frightened					
1000ms	2, 84	1.7	0.09	0.19	.039
2000ms	2, 84	2.4	0.24	0.10	.054
Happy					
1000ms	2, 84	0.58	0.63	0.56	.014
2000ms	2, 84	0.04	0.06	0.96	.001
Surprised					
1000ms	2, 84	0.24	0.04	0.79	.006
2000ms	2, 84	1.56	0.12	0.22	.036
Sad					
1000ms	2, 84	0.47	0.77	0.62	.011
2000ms	2, 84	2.78	3.95	0.07	.062

Table 6.8

Paired samples t-test results for frequency of response for each face type when interpreting the neutral face

Face 1	Face 2	*t*	df	*p*
Aggressive	Disgusted	4.25	86	.000***
	Frightened	8.6	86	.000***
	Happy	0.04	86	0.971
	Surprised	7.79	86	.000***
	Sad	-4.89	86	.000***
Disgusted	Frightened	5.74	86	.000***
	Happy	-3.61	86	.001**
	Surprised	5.15	86	.000***
	Sad	-9.32	86	.000***
Frightened	Happy	-7.06	86	.000***
	Surprised	-0.67	86	0.508
	Sad	-13.64	86	.000***
Happy	Surprised	7.11	86	.000***
	Sad	-4.27	86	.000***
Surprised	Sad	-13.1	86	.000***

* $p<.05$, ** $p<.01$, *** $p<.001$.

Reaction Time: Mean reaction times were analysed with three-way repeated measures ANOVAs for group by face type by stimulus duration (see Table 6.9 for means and standard deviations).

Table 6.9

Mean (and standard deviations) reaction times on the emotion recognition task for the three participant groups

Face Type	Group			Total
	Impulsive Aggressive	Premeditated Aggressive	Control	
Aggressive				
1000ms	2211.24 (845.55)	2383.61 (1101.13)	2773.74 (1602.79)	2449.55 (1228.53)
2000ms	1989.27 (1084.74)	2194.76 (772.33)	2461.26 (1081.25)	2204.09 (1025.79)
Total	2100.25 (860.24)	2304.10 (787.44)	2617.50 (1176.70)	2330.16 (980.49)
Disgusted				
1000ms	2147.30 (792.28)	2208.84 (1026.03)	2304.68 (848.57)	2217.26 (864.56)
2000ms	1948.24 (773.18)	2109.13 (874.75)	2101.85 (825.32)	2041.11 (810.90)
Total	2047.77 (698.48)	2171.67 (832.54)	2203.26 (708.11)	2132.72 (732.31)
Frightened				
1000ms	2298.14 (1149.74)	2494.60 (850.00)	2862.81 (1280.45)	2543.09 (1148.70)
2000ms	2127.09 (851.01)	2355.17 (836.43)	2344.82 (847.00)	2258.74 (843.28)
Total	2212.62 (922.84)	2489.93 (836.04)	2603.82 (939.06)	2417.64 (913.93)
Happy				
1000ms	1571.72 (356.18)	1844.72 (560.28)	1835.38 (639.53)	1730.36 (530.69)
2000ms	1668.32 (625.63)	1789.80 (404.44)	1871.06 (567.20)	1768.70 (559.26)
Total	1620.02 (440.41)	1803.99 (430.91)	1853.22 (508.88)	1746.95 (469.76)
Neutral				
1000ms	2955.53 (1182.57)	3474.48 (2080.79)	3837.87 (2013.88)	3390.05 (1764.13)
2000ms	2979.72 (1508.19)	3027.43 (1372.27)	3472.16 (2219.81)	3163.15 (1756.77)
Total	2967.63 (1155.64)	3196.73 (1468.49)	3655.02 (1866.92)	3262.59 (1523.19)
Surprised				
1000ms	2047.77 (853.34)	2072.28 (560.13)	2079.65 (689.60)	2064.88 (726.33)
2000ms	1847.25 (734.95)	2061.36 (724.08)	2255.94 (958.93)	2042.10 (827.66)
Total	1947.51 (727.55)	2142.98 (649.93)	2167.79 (723.95)	2072.90 (707.07)

Table 6.9 continued

Face Type	Group			Total
	Impulsive Aggressive	Premeditated Aggressive	Control	
Sad				
1000ms	2245.36 (1050.87)	2612.95 (1783.10)	2562.12 (1133.66)	2445.62 (1287.06)
2000ms	1918.09 (692.94)	2124.99 (1130.28)	2454.14 (1239.50)	2155.60 (1035.25)
Total	2081.72 (722.92)	2591.77 (1706.44)	2508.13 (1116.57)	2357.74 (1179.40)
All Face Types				
1000ms	2211.01 (656.09)	2490.23 (906.21)	2608.04 (876.55)	2418.55 (81313)
2000ms	2068.28 (682.44)	2281.44 (720.89)	2423.03 (739.81)	2244.51 (720.89)

Analyses for reaction time revealed a significant main effect of face type, $F(4, 306) = 42.22$, $MSE = 6.1$, $p<.001$, $\eta^2 = .337$. Paired samples *t*-tests indicated that participants responded significantly faster to the happy face type than the other six face types. Surprised and disgusted faces were responded to significantly faster than the aggressive, frightened, neutral and sad faces. Aggressive, sad and frightened faces were responded to significantly faster than neutral faces. See Table 6.10 for results of the *t*-tests.

A significant effect of stimulus duration was found, $F(1, 83) = 17.99$, $MSE = 9039449.44$, $p<.001$, $\eta^2 = .178$. Participants responded significantly faster when faces were presented for 2000ms than 1000ms. No other significant main effects or interactions were found for reaction times on the emotion recognition task.

Table 6.10

Paired samples t-test results for reaction time for each face type

Face 1	Face 2	*t*	df	*p*
Aggressive	Disgusted	2.85	86	.005**
	Frightened	-1.08	86	.283
	Happy	7.17	86	.000***
	Neutral	-7.88	86	.000***
	Surprised	3.39	86	.001**
	Sad	-0.23	86	.817
Disgusted	Frightened	-3.40	86	.001**
	Happy	6.47	86	.000***
	Neutral	-8.74	86	.000***
	Surprised	0.84	86	.404
	Sad	-2.02	86	.046*
Frightened	Happy	9.03	86	.000***
	Neutral	-6.42	86	.000***
	Surprised	4.86	86	.000***
	Sad	0.49	86	.000***
Happy	Neutral	-11.33	86	.000***
	Surprised	-5.13	86	.000***
	Sad	-5.19	86	.000***
Neutral	Surprised	8.37	86	.000***
	Sad	5.85	86	.000***
Surprised	Sad	-2.61	86	.011*

* $p<.05$, ** $p<.01$, *** $p<.001$.

6.6.3 Aggression rating task

Participants' raw aggressiveness ratings and reaction times were collated for the five face types, and means and standard deviations calculated for each group.

Means were analysed with three-way repeated measures ANOVAs for group by face type by stimulus duration.

Response: Analyses for response revealed a significant effect of face type, F(3, 283) = 509.22, MSE = 172.9, $p<.001$, $\eta^2 = .858$. Paired-samples *t*-tests indicated that participants rated aggressive faces as significantly more aggressive than frightened, $t(86) = 27.53$, $p<.001$, happy, $t(86) = 33.44$, $p<.001$, and neutral faces, $t(86) = 27.11$, $p<.001$, disgusted faces as significantly more aggressive than frightened, $t(86) = 23.31$, $p<.001$, happy, $t(86) = 28.92$, $p<.001$, and neutral faces, $t(86) = 21.94$, $p<.001$, frightened faces as significantly more aggressive than happy faces, $t(86) = 9.26$, $p<.001$, and neutral faces as significantly more aggressive than frightened, $t(86) = -5.17$, $p<.001$, and happy faces, $t(86) = -14.81$, $p<.001$. No other significant main effects or interactions were found for response on the aggression rating task (see Table 6.11 for means and standard deviations). These findings support the validity of the face stimuli to be used in the current investigation.

To further investigate group differences relevant to the research hypothesis, separate univariate ANOVAs were completed for each face type. A significant effect of group was found for the neutral face type, F(2, 86) = 3.17, MSE = 0.71, $p = .047$, $\eta^2 = .070$. Post hoc Tukeys indicated that the premeditated-aggressive groups rated the neutral faces as significantly more aggressive than did the control group ($p<.05$). No effect of group was found for the aggressive, F(2, 86) = 2.1, MSE = 0.72, $p = 0.13$, $\eta^2 = .048$, disgusted, F(2, 86) = 0.56, MSE = 0.24, $p = 0.57$, $\eta^2 = .013$, frightened, F(2, 86) = 0.37, MSE = 0.09, $p = 0.69$, $\eta^2 = .009$, or happy face types, F(2, 86) = 1.03, MSE = 0.003, $p = 36$, $\eta^2 = .024$.

Table 6.11

Mean (and standard deviations) responses for the three participant groups on the aggressive rating task

Face Type	Group			Total
	Impulsive Aggressive	Premeditated Aggressive	Control	
Aggressive				
1000ms	3.19 (0.70)	3.17 (0.64)	2.85 (0.62)	3.07 (0.67)
2000ms	3.22 (0.77)	3.21 (0.62)	3.01 (0.68)	3.14 (0.70)
Total	3.21 (0.67)	3.19 (0.53)	2.93 (0.51)	3.11 (0.59)
Disgusted				
1000ms	3.06 (0.75)	2.99 (0.72)	2.87 (0.60)	2.97 (0.69)
2000ms	3.10 (0.73)	2.99 (0.78)	2.95 (0.80)	3.03 (0.76)
Total	3.08 (0.67)	2.99 (0.66)	2.91 (0.64)	3.00 (0.65)
Frightened				
1000ms	1.56 (0.45)	1.49 (0.54)	1.47 (0.54)	1.51 (0.50)
2000ms	1.50 (0.53)	1.40 (0.55)	1.41 (0.49)	1.44 (0.52)
Total	1.53 (0.47)	1.45 (0.52)	1.44 (0.48)	1.48 (0.48)
Happy				
1000ms	1.02 (0.70)	1.04 (0.13)	1.01 (0.04)	1.02 (0.08)
2000ms	1.01 (0.34)	1.03 (0.09)	1.02 (0.06)	1.02 (0.06)
Total	1.01 (0.04)	1.03 (0.08)	1.01 (0.04)	1.02 (0.06)
Neutral				
1000ms	1.86 (0.56)	2.04 (0.68)	1.62 (0.41)	1.82 (.057)
2000ms	1.69 (0.46)	1.89 (0.63)	1.64 (0.49)	1.72 (0.52)
Total	1.77 (0.45)	1.96 (0.57)	1.63 (0.41)	1.77 (0.48)

Reaction Time: Overall means and standard deviations for reaction times on the aggression rating task are shown in Table 6.12.

Table 6.12

Mean (and standard deviations) reaction times for the three participant groups on the aggressive rating task

Face Type	Group			Total
	Impulsive Aggressive	Premeditated Aggressive	Control	
Aggressive				
1000ms	921.03 (504.76)	956.21 (654.68)	1294.33 (568.25)	1058.65 (586.55)
2000ms	661.10 (320.94)	1155.53 (920.23)	1072.19 (590.20)	927.88 (643.78)
Total	791.06 (382.91)	1055.87 (660.37)	1183.26 (519.49)	993.27 (534.50)
Disgusted				
1000ms	835.57 (529.29)	1175.83 (703.64)	1288.45 (510.67)	1077.78 (601.18)
2000ms	838.71 (649.62)	1038.83 (930.91)	1012.63 (550.32)	949.29 (699.29)
Total	837.14 (532.45)	1107.33 (770.41)	1150.54 (481.46)	1013.53 (597.20)
Frightened				
1000ms	725.26 (534.71)	9328.78 (532.85)	1015.99 (516.14)	879.50 (537.85)
2000ms	617.25 (442.95)	790.89 (605.46)	959.52 (939.07)	779.18 (535.84)
Total	671.25 (443.56)	864.40 (535.56)	987.23 (476.20)	829.23 (493.49)
Happy				
1000ms	470.20 (230.56)	694.97 (814.51)	580.58 (216.29)	565.10 (454.81)
2000ms	470.20 (301.60)	523.90 (216.93)	591.54 (258.68)	525.91 (269.72)
Total	470.56 (242.70)	609.43 (485.39)	586.06 (186.94)	545.50 (310.66)
Neutral				
1000ms	732.80 (423.29)	804.38 (556.64)	1005.99 (462.13)	845.10 (482.80)
2000ms	570.12 (259.15)	852.04 (513.43)	972.69 (563.54)	780.23 (479.81)
Total	651.46 (308.90)	828.21 (506.50)	989.34 (469.48)	812.67 (442.96)
All Faces				
1000ms	736.97 (342.60)	914.03 (586.58)	1037.07 (331.37)	885.23 (429.71)
2000ms	631.62 (313.91)	872.24 (509.51)	921.71 (385.87)	792.50 (412.97)
Total	684.29 (317.77)	893.14 (528.24)	979.39 (345.11)	838.86 (406.92)

The analyses revealed a significant main effect of face type, F(4, 305) = 35.11, MSE = 6758816.87, $p<.001$, $\eta^2 = .295$. Paired samples *t*-tests indicated that participants responded significantly faster to happy faces than aggressive faces, $t(86) = 9.05$, $p<.001$, disgusted faces, $t(86) = 9.98$, $p<.001$, frightened faces, $t(86) = 7.17$, $p<.001$, and neutral faces, $t(86) = -7.17$, $p<.001$. Neutral faces were responded to significantly faster than aggressive faces, $t(86) = 3.94$, $p<.001$, and disgusted faces, $t(86) = 4.63$, $p<.001$. Frightened faces were responded to significantly faster than aggressive faces, $t(86) = 3.39$, $p = .001$, and disgusted faces, $t(86) = 3.72$, $p<.001$.

A significant main effect of stimulus duration was also found, F(1, 84) = 13.3, MSE = 1604582.14, $p<.001$, $\eta^2 = .137$. Participants responded significantly faster to the faces at the 2000ms stimulus duration than the 1000ms stimulus duration. The main effect of group was also significant, F(1, 84) = 4.92, MSE = 7467236.68, $p = 0.10$, $\eta^2 = .105$. Post hoc Tukey's indicated that control participants took significantly longer to respond to the faces than the impulsive-aggressive group. The premeditated-aggressive group did not differ from the impulsive-aggressive or control groups. These effects were modified by a significant face type by stimulus duration by group interaction, F (7, 84) = 3.12, MSE = 380480.69, $p = .003$, $\eta^2 = 069$ (see Figures 6.2 and 6.3). Separate ANOVAs were then conducted for each stimulus duration.

At the 1000ms stimulus duration, a significant main effect of face type was found, F(4, 305) = 28.39, MSE = 3891266, $p<.001$, $\eta^2 = .253$. Follow-up paired-samples *t*-tests indicated that participants responded significantly faster to the happy face than the aggressive, disgusted, frightened and neutral faces. Neutral and frightened faces were responded to significantly faster than the aggressive and disgusted faces (*p*s<.05). See Table 6.13 for results of the *t*-tests. The main effect of group was also significant, F(2, 84) = 4.31, MSE = 3698064, $\eta^2 = .093$. Tukey's post

hoc tests indicated that the control group took significantly longer to respond to the faces than the impulsive-aggressive group. These two significant main effects were qualified by a significant face type by group interaction, $F(7, 305) = 2.19$, $MSE = 299744.61$, $p = .033$, $\eta^2 = .049$. Follow-up univariate ANOVAs for each face type indicated that the impulsive-aggressive group responded significantly faster than the control group to the aggressive faces, $F(2, 86) = 3.98$, $MSE = 1280077.27$, $p = .022$, $\eta^2 = .084$, and disgusted faces, $F(2, 86) = 5.50$, $MSE = 1798164.35$, $p = .006$, $\eta^2 = .116$. No significant group differences were found for the frightened, $F(2, 86) = 2.64$, $MSE = 734416.52$, $p = .059$, $\eta^2 = .000$, happy, $F(2, 86) = 1.7$, $MSE = 346730.73$, $p = .188$, $\eta^2 = .039$, or neutral faces, $F(2, 86) = 2.8$, $p = .066$, $\eta^2 = .063$.

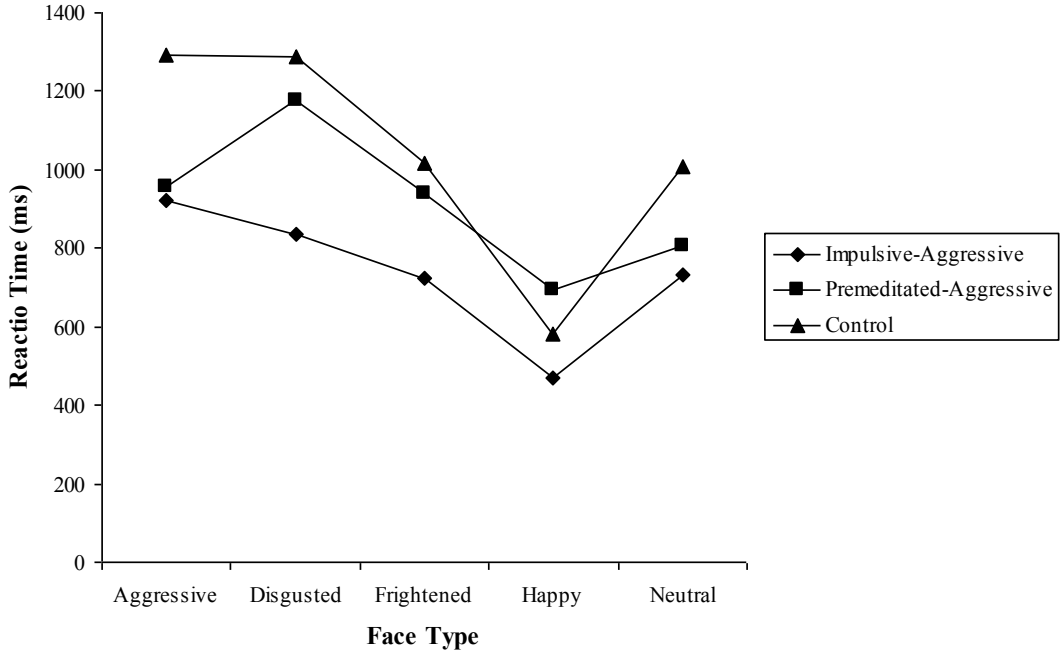

Figure 6.2. Reaction times for each face type by each group at the 1000ms stimulus duration.

Table 6.13

Paired samples t-test results for face type at the 1000ms stimulus duration

Face 1	Face 2	*t*	df	*p*
Aggressive	Disgusted	-0.39	86	.699
	Frightened	3.01	86	.003**
	Happy	8.04	86	.000***
	Neutral	3.92	86	.000***
Disgusted	Frightened	3.44	86	.001**
	Happy	9.71	86	.000***
	Neutral	4.45	86	.000***
Frightened	Happy	6.09	86	.000***
	Neutral	0.62	86	.536
Happy	Neutral	-6.09	86	.000***

* $p<.05$, ** $p<.01$, *** $p<.001$.

At the 2000ms stimulus duration, a significant main effect of face type was found, $F(3, 289) = 16.83$, $MSE = 3086966.82$, $p < .001$, $\eta^2 = .167$. Paired samples *t*-tests indicated that participants responded to happy faces significantly faster than the other face types. Neutral and frightened faces were responded to significantly faster than aggressive and disgusted faces. See Table 6.14 for results of the *t*-tests. A significant main effect of group was also found $F(1, 84), = 4.95$, $MSE = 3866686.46$, $\eta^2 = .105$. Post hoc Tukeys indicated that the control group took significantly longer to respond to the faces than the impulsive-aggressive group. The face type by group interaction was not significant at the 2000ms stimulus duration.

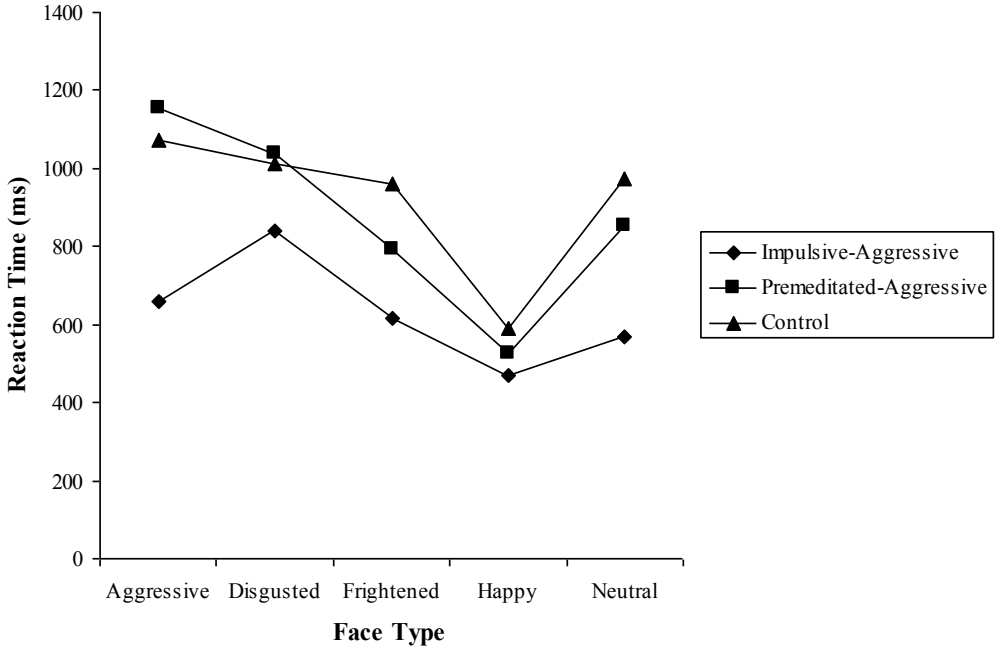

Figure 6.3. Reaction times for each face type by each group at the 2000ms stimulus duration.

Table 6.14

Paired samples t-test results for face type at the 2000ms stimulus duration

Face 1	Face 2	*t*	df	*p*
Aggressive	Disgusted	-0.31	86	.761
	Frightened	2.58	86	.012*
	Happy	6.03	86	.000***
	Neutral	2.23	86	.029*
Disgusted	Frightened	2.67	86	.009**
	Happy	6.26	86	.000***
	Neutral	2.73	86	.008**
Frightened	Happy	5.13	86	.000***
	Neutral	-0.02	86	.983
Happy	Neutral	-5.28	86	.000***

* $p<.05$, ** $p<.01$, *** $p<.001$.

6.6.4 Wechsler Adult Intelligence Scale – Third Edition

Univariate ANOVAs revealed no significant differences between the participant groups on Vocabulary, $F(2, 86) = 1.7$, $MSE = 6.92$, $p = .189$, $\eta^2 = .039$, or Digit Span, $F(2, 86) = .035$, $MSE = .082$, $p = .966$, $\eta^2 = .001$. Mean scores for both tasks are shown in Table 6.15.

Table 6.15

Means (and standard deviations) for Vocabulary and Digit Span for the three participant groups

	Impulsive-Aggressive	Premeditated-Aggressive	Control	Total
Vocabulary	10.71 (2.04)	11.59 (2.58)	10.63 (1.45)	10.91 (2.03)
Digit Span	10.40 (1.83)	10.50 (1.41)	10.40 (1.22)	10.43 (1.52)

6.7 Discussion

The purpose of this study was to investigate whether impulsive-aggressive and/or premeditated aggressive individuals would demonstrate impairment in the interpretation of facial expressions of affect. This study also investigated possible hostile attribution biases among this population by examining the interpretations each group made of the neutral faces they were exposed to and their ratings of aggression towards these faces.

6.7.1 Emotion recognition task

On the emotion recognition task, it was hypothesised that the impulsive-aggressive group would display a „hostile attribution bias'; a tendency to view neutral

expressions as hostile. The results did not support the hypothesis, instead indicating that the three groups were most likely to attribute an emotion seemingly irrelevant to aggression (most often „sad').

This result is analogous to that of Hoaken et al. (2007) for their non-violent offender and control groups. Their violent offenders, however, most often responded with „disgust'. It is difficult to draw direct comparisons between the current findings and that of Hoaken et al., however, given the lack of detail regarding characteristic aggression in their sample. While it could be assumed that their offender sample, both violent and non-violent, would present with aggression, this was not measured specifically. Therefore, a preliminary theory to account for these differences could be that individuals displaying severe antisocial behaviour more broadly present with a deficit in emotion recognition. In contrast, the present sample, who may not present with such significant antisocial behaviours and instead are characterised by high levels of trait aggression more specifically, may not have impairments in this area.

Hoaken et al. (2007) also found that the violent offender group was significantly poorer at the interpretation of facial expressions of emotion than either non-violent offenders or controls. In the present study, all groups made significantly more errors when interpreting the disgusted and frightened faces than the other emotional expressions. Within this analysis, the only significant group difference showed that the frequency with which the premeditated-aggressive individuals rated the neutral faces as frightened was significantly greater than the control group.

Neuroimaging studies have implicated the amygdala in the processing of fearful faces. Similarly, psychopathy has been linked to amygdala deficits (Blair, 2003a) and has been linked to impairments in the recognition of fearful facial expressions (Blair et al., 2004). The finding that the premeditated-aggressive

individuals showed a bias towards interpreting fear in neutral faces suggests possible amygdala dysfunction in this population. This hypothesis is supported by the proposed relationship between psychopathy and the expression of premeditated-aggression. One important distinction between the study by Blair et al. and the current study, however, is that Blair et al. used an emotional expression multimorph task consisting of facial images morphed from an emotional expression to neutral affect, while the present study utilised a rating task. That is, there are differences in the ability to distinguish an emotion from a neutral expression and the ability to provide a single categorical label to the stimulus. Furthermore, the psychopathic population made a significant greater number of errors when interpreting the fearful face, while the current premeditated-aggressive group did not make such errors. While these preliminary findings do indeed suggest a relationship between premeditated-aggression, psychopathy, and the interpretation of fearful facial expressions, more detailed analyses are required before more concrete theories can be postulated.

Patients with orbitofrontal lesions have been shown to have impairments in the recognition of anger and disgust (Blair & Cipolotti, 2000). Furthermore, increases in orbitofrontal activity measured with functional imaging have been found in response to angry expressions (Blair et al., 1999), and to the actual induction of anger (Dougherty et al., 1999; Kimbrell et al., 1999). On the basis of this research, it was proposed that the impulsive-aggressive individuals, due to orbitofrontal dysfunction, would be impaired in the recognition of angry faces. Furthermore it was suggested that impulsive-aggressive individuals would be primed to perceive negative emotion in neutral situations, which would explain why impulsive-aggressive individuals seem to be easily provoked into negative interactions and conflicts with others. Indeed, Best et al. (2002) found that individuals with Intermittent Explosive Disorder, a psychiatric

disorder characterised by impulsive-aggression, were biased to label neutral faces with „disgust' and „fear', both negatively valenced expressions. This result was not replicated in the current study.

Decoding facial expressions of emotion is likely to be involved in normal social and emotional responses to other individuals. Faces convey reinforcing signals, and the orbitofrontal cortex plays a vital role in the learning of the reinforcing signals used in social communication (Hornak et al., 1996). The results of this study indicate that impulsive- and premeditated-aggressive individuals do not generally have impairment in the interpretation of emotions in others. This finding is in contrast with Hornak et al. who found a strong association between face decoding deficits and subjective and objective reports of emotional changes in behaviour. That is, patients with damage to the ventral frontal lobe who had greater alteration in emotional experience had poorer performance on facial expression identification. However, this result also indicates that those with minimal emotional change were not as impaired on this task. Thus, it may be suggested that given the current sample, who while having high levels of trait aggression are continuing to function adaptively within the community and may only present subclinical impairment, do not demonstrate such severe deficits in emotion recognition. In support of this hypothesis, the patients in Hornak et al.'s study suffered significant damage to the ventral region of the prefrontal cortex either through head injury or cerebrovascular accident, while the current sample had no history of head injury.

A further suggestion lies in the presence of trait aggression in comparison to subjective „emotional change' which may or may not have involved an aggressive component. Thus it can be suggested that emotion interpretation deficits may not be

present in aggressive individuals specifically, but in those individuals displaying antisocial or emotional changes more broadly.

The identification of various emotional expressions may also be related to the issue of task difficulty. That is, selective impairments in recognising certain emotions may be due to that fact that some emotional expressions may be more difficult to discriminate than others. This may be because some expressions are configurally ambiguous, are more complex, or may make specific demands on the perceptual system (Adolphs, 2002a). Rapcsak et al. (2000) used a labelling task to investigate the issue of task difficulty in a sample of brain-damaged subjects and normal controls. The authors reported that recognition of fear was less accurate than recognition of other emotions even in normal subjects. This was also demonstrated in the current study, with frightened faces being the expression interpreted with least accuracy.

Happiness could be viewed as a superordinate category and the other basic negatively valenced emotions as subordinate categories of the superordinate category of unhappy (Adolphs, 2002a). This may help to explain why expressions of happiness were interpreted with significantly more accuracy than the relatively negatively valenced emotions including sad, aggressive, disgusted, and surprised. In Rapcsak et al.'s (2000) study, they found that the errors typically made by brain-damaged subjects were to mistake a negatively valenced emotion for another negatively valenced emotion but not for happiness. The errors in interpreting frightened faces occurred despite longer reaction times. The results indicate that those faces interpreted with most accuracy were also responded to significantly faster. This provides additional evidence for the above argument that certain faces of emotional affect are interpreted with greater ease.

6.7.2 Aggression rating task

Contrary to the hypothesis, the premeditated-aggressive group rated the neutral faces as significantly more aggressive than did the control group. This finding is not consistent with previous research demonstrating hostile attributional biases among impulsive-aggressive children in their attributions of peer's intentions in provocation situations (Dodge & Coie, 1987). Thus the misattribution of hostile intent in adults appears to be associated with elevated levels of premeditated-aggression rather than impulsive-aggression. These interpretations occurred despite variations in the duration of stimulus presentation. This suggests that premeditated-aggressive individuals have a negative attribution bias that affects their ability to interpret ambiguous social situations correctly such that they interpret neutral situations negatively. Indeed, the work on hostile attributional bias (Dill, Anderson & Deuser, 1997; Epps & Kendall, 1995; Hall & Davidson, 1996) indicates that hostile individuals do indeed see others as hostile.

It is proposed then that the processing mechanism responsible for the display of premeditated-aggression may be a hostile attribution bias. In terms of the described social information-processing model, it appears that premeditated-aggressive individuals have a tendency to utilise self-schemas rather than the presented cues provided. As the self-schemas of aggressive individuals may be more hostile than those of non-aggressive individuals, a reliance on self-schemas could lead aggressive individuals to make errors of presumed hostility (Dodge, Price, Bachorowski & Newman, 1990). This is consistent with the results of Dodge and Tomlin (1987) who found that a reliance on self-schemas is related to erroneous interpretations of the social environment. Similarly, Hall (2006) found a positive correlation between self-reported aggression and errors in perceiving aggression in others when it did not exist.

These findings support the possibility that the way in which social information is processed may mediate the expression of aggression.

People develop a perceptual set which establishes one's expectations and guides one's interpretation of social stimuli and situations. Thus, on the basis of the current findings, it can be argued that premeditated-aggressive individuals are more likely to utilise these aggressively-themed self-schemas when interpreting socially ambiguous situations. These hypotheses are in line with those of Grafman and colleagues who argued that knowledge stored in the prefrontal cortex plays a managerial role in the control of behaviour, taking the form of thematic understanding, planning, and understanding social rules (Grafman, 1994; Grafman et al., 1996). Social schema knowledge is thought to inhibit abnormal behaviour, and in this way, it would be expected that lesions to the prefrontal cortex would impair the ability to access this knowledge, biasing the regulation of behaviour away from social rules, toward environmental responsiveness, making aggressive behaviour more likely. Thus, if premeditated-aggressive individuals are perceiving hostility in socially ambiguous situations, they are more likely to respond in a similarly aggressive manner. This aggressive reaction will not be impulsive, but a planned, instrumental act of aggression against another who has been perceived to harm them.

In contrast to expectations, characteristic impulsivity does not cause impulsive-aggressive individuals to make rapid decisions, facilitating the cognitive mediation between their self-schemas and their response. Rather, they accurately interpreted the information presented to them. This type of bottom-up processing involving the integration of relevant environmental cues thus leads to more accurate interpretations than will schema-based processing that ignores these cues. Thus, given that impulsive-aggressive individuals do not present with the hypothesised social

information processing deficit, other factors, such as executive functioning deficits must contribute to the expression of impulsive-aggressive.

6.7.3 Personality measures

On the impulsivity-venturesomeness subscale of the I7 Impulsivity Questionnaire, there was a trend indicating higher scores among the impulsive-aggressive group in comparison to the control group. The impulsive-aggressive group also had significantly higher impulsivity scores than the premeditated-aggressive and control groups. This finding is comparable to that found in Study 1 and provides further evidence of specific personality pathology among impulsive-aggressive individuals. This characteristic impulsivity may cause an inability to inhibit aggressive responses in provocative social situations. In contrast, premeditated-aggressive individuals, without trait impulsivity, can inhibit spontaneous acts of aggression, instead planning when and where their aggression would be displayed.

On the empathy subscale of the I7 Impulsivity Questionnaire, the premeditated-aggressive group had significantly lower scores than the impulsive-aggressive and control groups. This result, while in contrast to those findings from Experiment 1, would be expected given the lack of remorse and subsequent justification which characterises their aggressive actions. This result also provides further evidence for the proposed premeditated-aggression-psychopathy relationship (e.g., Cornell et al., 1996). Further research delineating this relationship is needed, however, given the lack of consistency in the I7 results across the current studies.

6.7.4 The link between emotion recognition and aggression

Green (1998) stated that behaviour in situations of interpersonal conflict is guided by cognitive representations of events. Therefore, the incidence of aggression

is to a large extent a function of how social information is processed. Aggressive facial expressions are argued to act as social cues to initiate response reversal, causing the observer to either suppress the current response or to select an alternative response (Blair & Cipolotti, 2000) and in this way, trigger an inhibition of aggressive behaviour. However, in this instance, premeditated-aggressive individuals do not use this type of reinforcing stimuli to stop an aggressive reaction; rather it may encourage an aggressive response due to their past experiences. That is, through a history of positive reinforcement from aggressive interactions, premeditated-aggressive individuals have learnt that this response can be rewarding. In contrast, in non-aggressive individuals, schemas activated in the frontal lobes would be used as a means to inhibit aggressive responding, as they may have encountered negative aggressive reactions with others.

The experience and expression of emotion are correlated (Rosenberg & Ekman, 1994). Thus, production of emotional expressions can lead to changes in emotional experience through influencing the feeling and autonomic correlates of the emotional states (Levenson et al., 1990). Viewing facial expressions results in expressions on one's own face that may mimic the expression shown on the other individual's face (Hess & Blairy, 2001). Viewing the facial expression of another can thus lead to changes in one's own emotional state which in turn could result in a change of feeling (Wild, Erb & Bartels, 2001). Wild et al. argued that this process is fast and automatic, and provides a basis for one's own reactions. With regard to premeditated-aggression, it can therefore be argued that the interpretation of neutral expressions as aggressive leads to a consequent aggressive emotion in the individual. Rather than reacting impulsively to this stimuli, however, such individuals are able to

inhibit this response initially and instead plan and carry out an aggressive response at a later time.

6.7.5 Conclusion

The findings of the current study facilitate the understanding of how emotion recognition and hostile attributional biases may contribute to the expression of impulsive- and premeditated-aggression. The results suggest a tendency to attribute hostile intent in ambiguous social situations, coupled with low level empathy may contribute to the expression of premeditated-aggression. While premeditated-aggressive individuals do not necessarily make incorrect interpretations of various facial images of affect, they have a tendency to attribute greater levels of aggressiveness to neutral expressions. This suggests that premeditated-aggressive individuals may interpret benign social interactions as hostile and consequently retaliate in a similarly aggressive manner.

Given the lack of deficits present in the impulsive-aggressive group, the findings suggest distinct cognitive processes between individuals who are characteristically impulsive-aggressive and those who are characteristically premeditated-aggressive. These findings have important implications for our understanding of social information processing in aggressive individuals as it appears that premeditated-aggressive individuals demonstrate cognitive biases which are distinct from those which characterise impulsive-aggressors.

These results, while only providing preliminary support for the role of social information processing in premeditated-aggression, suggest that the expression of premeditated-aggression may be due to deficits at the encoding and interpretation stages of social information processing. This deficit results in the generation and

enactment of socially unacceptable responses. In order to add further weight to this proposed relationship, the subsequent stages of Crick and Dodge's (1994) social information processing model should be investigated in this population, including the goal selection, response generation, and evaluation of the response.

This finding also has important implications for our understanding of how perceptions of and social interactions with others impact upon the demonstration of aggressive behaviour. A predisposition towards an aggressive response may occur because the person perceives the environmental cues that appear to warrant this type of response. Thus, such individuals would benefit from assistance in reassessing and modifying what is being perceived and what is reality.

This study explored the role of the orbitofrontal cortex in the expression of impulsive- and premeditated-aggression through an investigation of emotion recognition and attribution of emotion towards neutral faces of emotion. While such abilities may contribute to the propensity for premeditated-aggression, the following study will attempt to further delineate the role of the orbitofrontal cortex in mediating subtypes of aggressive behaviour through an analysis of inhibitory, response reversal, and decision-making abilities in this population.

Chapter 7

Study 3: Inhibition, Response Reversal, and Decision-Making

The purpose of this study was to test whether subjects who are impulsive-aggressive versus premeditated aggressive differ on measures of inhibition, response reversal and decision-making.

7.1 Inhibition

The notion of impulsiveness is understood as a lack of response inhibition. Response inhibition can broadly be defined as the process by which a prepotent, routine, or dominant response is deliberately withheld (Hampshire, Chamberlain, Monti, Duncan & Owen, 2010). Inhibitory deficits thus involve an inability to suppress or withhold a previously rewarding response, and the behaviour appears impulsive (Bechara, 2004).

Inhibition is a critical component of behavioural control insofar as it enables us to overcome automatic or routine behaviours (Shallice & Burgess, 1993). So, on the one hand impulse control implies the ability to avoid risk and to curb excessive desire to seek sensation (Verdejo-Garcia, Lawrence & Clark, 2008), while on the other hand, implies cognitive operations that allow individuals to change behaviours in a dynamic fashion on the basis of information or feedback derived from monitoring ongoing behaviour (Kok, Ridderinkhof & Ullsperger, 2006). This latter capability has specifically been referred to as cognitive control. By setting goals, inhibiting habitual acts, and monitoring performance, cognitive control allows behavioural flexibility for one to function adaptively within changing environments and optimise goal-directed actions (Dalley, Cardinal & Robbins, 2004)

Inhibitory control is one of the executive control functions that determine how mental processes such as encoding, recognition, and retrieval, will work together in the performance of a task (Logan & Cowan, 1984). It is required in order to choose, construct, execute, and maintain optimal strategies for performing a task, and to inhibit strategies that become inappropriate. In addition to changes in the external environment, individuals might have to stop their action if they detect an error in their own performance. In these circumstances, deficient inhibitory control will lead to a greater likelihood that a response will be executed rather than withheld (Szatkowska et al., 2007).

Impulsive behaviour is mostly viewed as being a function of inhibitory control, that is, the ability to suppress undesirable response tendencies (Barkley, 1999). Whiteside and Lynam (2001) identified four personality facets conceived of as pathways to impulsive behaviour: (1) urgency, refers to the tendency to experience strong impulses, frequently under conditions of negative affect; (2) lack of premeditation, or the tendency to think and reflect on the consequences of an act before engaging in that act; (3) lack of perseverance, the ability to remain focused on a task that may be boring or difficulty; and (4) sensation seeking which is conceptualised as a tendency to enjoy and pursue activities that are exciting and to have an openness to try new experiences that may or may not be dangerous. Similarly, Barkley (1999) argued that impulsive behaviour in ADHD could be attributed to deficient inhibitory control. He distinguished three interrelated processes of behavioural inhibition: (1) the ability to withhold a prepotent response, (2) the ability to stop an ongoing response, and (3) interference control. Interference control is the ability to delay the decision to respond or prevent events and responses from interfering with an ongoing activity.

The definition of impulsivity thus incorporates a decreased sensitivity to negative consequences of behaviour and rapid, unplanned reactions to stimuli before complete processing of information. Moeller, Barratt, Dougherty, Schmitz and Swann (2001) incorporate these factors in their definition of impulsivity as "a predisposition toward rapid, unplanned reactions to internal or external stimuli without regard to the negative consequences of these reactions to the impulsive individual or to others" (p. 1784). The idea that impulsivity involves "rapid unplanned" action suggests that such behaviours occur before there is an opportunity to consciously consider the consequences of the act. This is distinct from impaired judgement or compulsive behaviour in which planning occurs before the behaviour.

It is important to draw a distinction between inhibitory control as a cognitive construct and impulsiveness as a behavioural construct. Impulsiveness is usually studied in terms of a response inhibition, that is, after establishing a habit to respond to a stimulus that predicts reward, there is a sudden change in the contingencies of the task (Bechara et al., 2000a). Cognitive inhibition, on the other hand, which can be seen as related to an inability to delay gratification, is a more complex form of disinhibited behaviour. There is a link between the two, but deficits in inhibitory control cannot account for all manifestations of impulsiveness. Cognitive impulsiveness can be observed in deficits in decision-making (as outlined in more detail below) (Barratt, 1994). In this way, it is a failure to delay gratification and evaluate the outcome of a planned action. In contrast, motor impulsiveness involves making a response before all of the necessary information has been obtained or the quick action without thinking (Evenden, 1999). Motor impulsiveness of an affective nature thus reflects an inability to inhibit a prepotent response that is affective, i.e., a prepotent reward response (Bechara, 2004).

7.1.1 Inhibition and the frontal lobes

Whereas the dorsolateral area appears to be primarily involved in the temporal integration of behaviours, such as working memory and planning, the orbital and ventromedial regions of the prefrontal cortex have a greater involvement in the inhibition of behaviours and control of emotional interferences (Roussy & Toupin, 2000). In line with this, inhibitory control throughout the lifespan appears to parallel the time course of prefrontal cortex maturation and decline (Dempster, 1992), and damage to the prefrontal cortex often leads to a behavioural „disinhibition syndrome' in which normal control over social behaviour is diminished (Fuster, 1996).

Lesion studies have provided equivocal support for the role of the orbitofrontal cortex in cognitive inhibition. Some authors argued that orbitofrontal lesions result in a loss of inhibitory control only in affective processing (Bechara, 2004; Bechara et al., 2000a, 2000b; Dias et al., 1996a) and contrasted this with an effect of dorsolateral prefrontal lesions on attentional inhibition and selection. On the other hand, orbitofrontal damage frequently results in impulsivity and disinhibition (Fuster, 1997), and patients with such damage are described as not being able to withhold responding regardless of whether the domain is affective or cognitive (Compton, 2003; Elliot, Dolan & Frith, 2000; Moscovitch & Winocur, 2002).

In support of the above theories, it has recently been proposed that inhibitory processes are organised in the prefrontal cortex according to different levels of abstraction and that different prefrontal areas may play a crucial role depending on whether lower-order or higher-order processes are required (Cools, Clark & Robbins, 2004; Roberts & Wallis, 2000). According to this view, the orbitofrontal cortex contributes to lower-order processes such as the inhibition and selection of specific

stimulus information, but not abstract task-rule information, which in turn is processed by the dorsolateral prefrontal cortex (Cools et al., 2004).

Using fMRI and EEG, Garavan, Ross, Murphy, Roche and Stein (2002) identified two systems involved in inhibition: errors were associated with medial activation incorporating the anterior cingulate and pre-supplementary motor area, while behavioural alteration subsequent to errors was associated with both the anterior cingulate and the left prefrontal cortex. The involvement of these two systems can be dissociated based on the relative difficulty of the response inhibition. Specifically, when ongoing target response speeds were relatively slow, response inhibition was executed by the right prefrontal system. This inhibitory system may instigate a more controlled inhibition, perhaps related to the role of the right dorsolateral prefrontal cortex in selecting the appropriate response over the prepotent response (Rowe, Toni, Josephs, Frackowiak & Passingham, 2000). The second inhibitory system, involving the anterior cingulate, was activated for inhibitions when ongoing response speeds were relatively fast, suggesting that this structure may be especially important in urgent inhibitions over faster or more automatic behaviours.

Berlin, Rolls and Kischka (2004) reported that patients with orbitofrontal cortex lesions performed more impulsively as shown by both self-report and cognitive/behavioural measures of impulsivity. This impulsiveness was specifically related to orbitofrontal damage, in that the non-orbitofrontal patients (who predominantly had damage to the dorsolateral prefrontal cortex) were not impaired on either measure. In the behavioural impulsivity task, orbitofrontal patients also made significantly faster responses than controls. This suggests that impulsivity may be at least partly due to a fast cognitive pace, which may lead to impatience or an inability to stop and think without acting. The authors also reported an associated between

inappropriate ‚frontal' behaviours and impulsivity in the orbitofrontal patients, suggesting that the patients may have been too impatient to wait for appropriate feedback and therefore failed to respond appropriately in social situations.

In order to account for the social cognition deficits observed in patients with frontal lobe dysfunction, Sahakian and colleagues proposed the ‚inhibition hypothesis' (Plaisted & Sahakian, 1997; Rahman, Sahakian, Hodges, Rogers & Robbins, 1999). The hypothesis proposes that the inability of a patient to suppress the response evoked by the stimulus prevents the patient from selecting an appropriate action plan. That is, orbitofrontal patients may act without giving themselves enough time to think about their behaviours and to modify them accordingly. Thus, the behaviour becomes dominated by the immediate emotional impact of the stimulus at hand.

7.1.2 Inhibition measures

Within the psychological literature, several behavioural models of impulsivity have been developed on the basis of findings from laboratory tasks used to measure impulsivity. These tasks fall into three broad categories: (1) punished and/or extinction paradigms, in which impulsivity is defined as the perseverance of a response that is punished or unrewarded; (2) reward-choice paradigms, in which impulsivity is defined as preference for a small immediate reward over a larger delayed reward; and (3) response disinhibition paradigms, in which impulsivity is defined either as making responses that are premature or an inability to withhold a response (Moeller et al., 2001).

The Stop Signal and Go/No-Go tasks have been widely used to investigate the behavioural and neural processes of response inhibition. For example, Aron et al.

(2003) found a high correlation between lesion volume within the right inferior frontal gyrus and inhibitory performance (r = .83). Rubia, Smith, Brammer and Taylor (2003) supported this result, finding that the right inferior prefrontal cortex was correlated with successful inhibition and failed inhibition was associated with activation in mesial frontopolar and bilateral inferior parietal cortices. More recently, Boehler, Appelbaum, Krebs, Hopf and Woldorff (2010), using conjunction analyses on fMRI data of successful and unsuccessful stop-trials, identified the lateral-inferior frontal and medial frontal cortical areas and the caudate nucleus. There are important differences between these two paradigms however; the most critical being that the Go/No-Go task is suited for measuring inhibition of prepotent responses, whereas the Stop Signal task measures inhibition of already initiated responses. The Stop Signal thus places a higher load on inhibitory control than the Go/No-Go Task (Boeker, Buecheler, Schroeter, & Gauggel, 2007).

Using fMRI, Chevrier, Noseworthy and Schachar (2007) found that there are distinct areas of the brain involved with the various aspects of inhibition and performance monitoring functions utilised during the Stop Signal Task. Namely, during the „Go' trials which involve response monitoring, activated right prefrontal and midline networks, while during „Stop' trials, involving response withdrawal activated right inferior frontal gyrus and basal ganglia. Furthermore, error detection invoked by failed inhibition activated dorsal anterior cingulate cortex and right middle frontal Brodmann's area 9.

Aron, Behrens, Smith, Frank and Poldrack (2007a) provided converging evidence for a frontal-subcortical network for response control, demonstrating increased activation of right pre-supplementary motor area, inferior frontal cortex, and subthalamic nucleus regions. Further support comes from Duann, Ide, Luo and Li

(2009) who argued that the inferior frontal cortex and pre-supplementary motor area play different roles in stop signal inhibition. Their results indicate that the inferior frontal cortex, an integral part of the ventral attention system, mediates attentional processing of the stop signal while the pre-supplementary motor area mediates response inhibition. The authors suggest that this greater inferior frontal cortex activity during stop- compared with go-trials may simply reflect attentional processing of the stop signal. Thus, by increasing activity in response to the stop signal, the inferior frontal cortex may serve to orient attention and resources to the stop process and, as a result, facilitate inhibition.

ERP research also relates inhibition to prefrontal activation, linking prefrontal activity reported in fMRI studies to the so-called Stop-N2 component in ERP research (e.g., Pliszka, Liotti & Woldorff, 2000; van Boxtel, van der Molen, Jennings & Brunia, 2001). Notably, this component has been demonstrated to be larger for successful compared with unsuccessful stoping (Schmajuk, Liotti, Busse & Woldorff, 2006).

The converging evidence from these various methodologies suggests that predominantly three brain areas are crucial for response inhibition in the Stop Signal Task: inferior frontal gyrus; medial prefrontal areas including the pre-supplementary motor area; and the basal ganglia (see Aron et al., 2007b, Chambers, Garavan & Bellgrove, 2009; Verbruggen & Logan, 2008 for reviews). The dominant view argues that, in response to a stop-stimulus, a signal from the inferior and/or medial frontal cortex is sent to the basal ganglia to cancel the motor program triggered by the go-stimulus (Aron et al., 2007a; Aron & Poldrack, 2006; Eagle et al., 2008; Ray et al., 2009). Interactions between the different parts of the basal ganglia and the associated

subthalamic nucleus produce a signal that is sent to the motor cortex via the thalamus, where the response is ultimately inhibited (Stinear, Coxon & Byblow, 2009).

7.1.3 Inhibition and aggression

Impulsivity can be divided into two distinct types: functional impulsivity refers to a rapid information processing style which leads to a tendency to make quick decisions when this strategy is appropriate to the situation; dysfunctional impulsivity is related to speedy and non-reflexive decisions, which have a negative consequence for the individual (Dickman, 1990). Dysfunctional impulsivity thus appears to be related to the inability to inhibit competing responses.

Although most people engage in impulsive behaviour at some time, high degrees of impulsivity have been linked to various forms of psychopathology and maladaptive behaviour. Previous research has shown higher levels of impulsivity among individuals with CD, personality disorders, and substance-abuse disorders (Moeller et al., 2001). Moreover, some authors consider impulsivity as the best predictor of adult antisocial and delinquent disorders (von Knorring & Ekselius, 1998).

Theorists have posited that impulsivity may be viewed as a personality characteristic that predisposes individuals to develop long-term, recidivistic antisocial behaviour. For example, Gorenstein and Newman (1980) proposed the existence of an underlying disinhibitory personality style that will be expressed as antisocial behaviour when it comes into conjunction with particular environmental risk factors. Further to this, Barratt (1994) suggested that some people are predisposed to respond to certain stimuli or situations with feelings of anger that may lead to an aggressive response. If such a predisposition is combined with a high level of impulsivity, then

the difficulty of inhibiting responses that is characteristic of impulsive individuals involves a lower response control, facilitating aggressive behaviour.

Impulsivity and low self-control have been shown to be consistent predictors of delinquency (Moffitt, Caspi, Harrington & Milne, 2002), and there is a well-documented relationship between impulsivity and antisocial behaviour among incarcerated men (Barratt et al., 1997b; Moeller et al., 2001; Wang & Diamond, 1999). Along with anger, hostility, and antisocial personality style, impulsivity has also been shown to be a strong predictor of institutional aggression, violence, and adjustment problems among incarcerated male offenders (Fornells, Capdevila & Andres-Pueyo, 2002; Wang & Diamond, 1999). More recently, Komarovskaya, Loper and Warren (2007) found that female inmates reported significantly higher impulsivity scores on a self-report measure than the normative sample and similar scores as psychiatric inpatients from the study of Patton, Stanford and Barratt (1995). Pratt and Cullen (2000) argued that the effect size for the relationship between low self-control and antisocial behaviour is one of the strongest correlates of criminal behaviour. The argument then follows that when anger exceeds inhibition, aggression is expressed behaviourally. Individuals with poorer inhibition will therefore express aggression more frequently. On the contrary, prefrontal effortful control enables long-term planning in which present impulses are inhibited in favour of possible long-term gains. As a result, people with low self-control have a relatively greater focus on immediate rewards compared to long-term consequences of their behaviour (MacDonald, 2008).

Few studies have investigated the aggression-impulsivity relationship directly. In one such study, LeMarquand et al. (1998) examined the relationship among tryptophan depletion, impulsivity and aggression in a sample of adolescent males

selected for an extensive history of aggressive behaviour. Aggressive individuals made more Go/No-Go „impulsive' errors than did non-aggressive individuals and had lower scores on tests of executive function. The authors suggested that impulsivity and executive function are correlated and underlie aggressive behaviour.

Stanford et al.'s (1997) study of neuropsychological performance and impulsive-aggression in college students supports this relationship. Specifically researchers noted that errors in processing and control of impulses indicative of executive functioning deficits were observed among impulsive-aggressive subjects. Hoaken et al. (2003) also attempted to test the relationship between executive functioning, aggression, and impulsivity. They found that executive functioning was related to both aggression and impulsivity as measured by the Go/No-Go task.

More recently, Vigil-Colet and Codorniu-Raga (2004) investigated the relationship between inhibitory deficits and aggression, and the role that impulsivity plays in the relationship. They found that inhibition deficits were specifically related to the anger scale of the BPAQ (Buss & Perry, 1992). Further to this, dysfunctional impulsivity was correlated with aggression, while functional impulsivity showed no significant relationship. Impulsive subjects also showed shorter reaction times and took less time to answer the questionnaires suggestive of non-reflexive strategies in this population which places more importance on speed than accuracy.

7.2 Response reversal

Reversal learning, by definition, involves a shift in responding from a stimulus that is no longer rewarding, to a previously unrewarded stimulus, while extinction refers to the normal reduction in behaviour when rewards are no longer given. An essential component of adaptive behaviour is the ability to learn the reward value of

stimuli, which can change according to an individual's circumstances and state. A previously rewarded stimulus may cease to be rewarding or even reverse its value and become punishing as a function of external changes in reinforcement contingencies.

7.2.1 Response reversal and the frontal lobes

There is growing evidence from functional neuroimaging studies, human lesion studies, and from neurophysiological and lesion studies in non-human primates that the ventral parts of the frontal lobe, which include the orbitofrontal cortex, play a crucial role in representing the reward and punishment value of stimuli and in rapidly learning or reversing associations between visual stimuli and rewards and punishments (Rolls, 1999, 2000, 2002). Following from this, the orbitofrontal cortex appears to play a critical role in the way in which this representation guides goal-directed behaviour (Rolls, 1999).

These hypotheses, and the role in particular of the orbitofrontal cortex in human behaviour, have been investigated in a number of studies in humans with damage to the ventral parts of the frontal lobe (the description „ventral' is given to indicate that there was pathology in the orbitofrontal or related parts of the frontal lobe, but not in the dorsolateral parts of it). Rolls and colleagues have studied stimulus-reward learning and the ability to reverse and/or extinguish responses that have been previously rewarded (Rolls et al., 1994). Control patients with damage elsewhere in the frontal lobes or in other regions did not show this deficit. The ventral frontal patients also made more errors on a similar extinction task in which the reward was no longer given. Interestingly, the patients were often able to verbally report that the contingencies had changed, but were unable to alter their behaviour appropriately. Fellows and Farrah (2003) supported these findings, demonstrating a selective

impairment on reversal learning in subjects with lesions of the ventromedial prefrontal cortex in comparison to a group with dorsolateral frontal lobe damage and normal controls.

In a more recent study, patients with lesions in different regions of the prefrontal cortex were tested on a probabilistic visual discrimination reversal test. On this task, it was necessary both to determine which feedback was crucial and to use this information appropriately to guide the choice of stimulus to maximise the reward obtained (Hornak et al., 2004). Patients with bilateral orbital/medial prefrontal lesions demonstrated a severe impairment on the test, whereas even large unilateral lesions, which included the orbital/medial region, had no such effect. Similarly, Berlin et al. (2004) reported that orbitofrontal patients performed poorer on a stimulus-reinforcement association reversal task than non-orbitofrontal cortex lesions controls and normal control participants.

Mitchell et al. (2006) reported on patient CL who had acquired psychopathy following an orbitofrontal cortex lesion. CL demonstrated intact stimulus-reinforcement based learning, but impaired reversal learning. In addition to this, CL showed severely disturbed social behaviour including high levels of impulsive-aggression. The authors reported that the patient resembled developmental psychopathy in the severity of their antisocial behaviour, violence, unconcern for the victims, and lack of insight toward the seriousness of their condition. However, it is important to note that such patients differ from individuals with psychopathy in that their offenses were impulsive with no history of premeditated-aggression.

Functional neuroimaging studies also implicate the orbitofrontal cortex in learning or reversing associations between visual stimuli and rewards or punishments (Cools, Clark, Owen & Robbins, 2002; O'Doherty et al., 2001). Rolls (2000)

demonstrated that there is a major visual input to many neurons in the orbitofrontal cortex, and that in many cases, these neurons represent the reinforcement association of visual stimuli. In addition to these neurons encoding the reward association of visual stimuli, other neurons in the orbitofrontal cortex detect non-reward, for example, responding when an expected reward is not obtained with a visual discrimination is reversed (for background, see Rolls, 2005). Kringelbach and Rolls (2003) used the faces of two different people, and if one face was selected then that face smiled, and if the other was selected, the face showed an angry expression. After good performance was acquired, there were repeated reversals of the visual discrimination task. They found that activation of a lateral part of the orbitofrontal cortex in the fMRI study was produced on the errors trials, that is, when the participant chose a face, and did not obtain the expected reward. Control tasks showed that the response was related to the error, and the mismatch between what was expected and what was obtained, in that just showing an angry face expression did not selectively activate this part of the lateral orbitofrontal cortex. The study revealed that the human orbitofrontal cortex is very sensitive to social feedback when it must be used to change behaviour.

7.2.2 Response reversal measures

Several investigators have developed behavioural paradigms to study the neural mechanisms underlying the behaviour of patients with lesions to the prefrontal cortex, with much of the work relying on two conceptually related measures: object reversal learning and extinction. The ability to update and correct behaviour on the basis of changes in emotional significance is critical to successful performance on such tasks (Rolls, 1999).

The Intra/Extra Dimensional Set Shift task (ID/ED) is used to gauge attentional set shifting and response reversal. The task assesses the participant's ability to maintain attention to different examples within the same dimension (ID stages) and then to shift attention to a previously irrelevant dimension (ED stages). This task thus assesses two dissociable abilities. Firstly, it assesses the ability to perform response reversals; the participant is initially rewarded for his/her behaviour then this stimulus is no longer associated with reward. Secondly, it assesses the ability of the participant to perform ED shifts; to shift their response set from one stimulus property to another, for example, shifting attention from the shape of the stimulus to lines (Rogers, Andrews, Grasby, Brooks & Robbins, 2000). Therefore, the acquisition of this new visual discrimination must occur in the face of an attentional-set established over the course of recent reinforcement history.

While the WCST is widely used to gauge the ability to shift cognitive set, and has been considered a cardinal measure of the function of the prefrontal cortex, the ID/ED Task allows the cognitive components of set shifting to be assessed independently and their contribution examined as the task increases in complexity (Fray et al., 1996). Accumulating evidence indicates that focal damage to the prefrontal cortex in both human neurological patients and experimental primates impairs ED shift learning (Dias et al., 1996a, 1996b, 1997; Owen et al., 1991, 1993). Dias et al. (1996a) reported that lesions of the orbitofrontal cortex produce impairments in the reversal learning, but not in the ED shift learning. In contrast, damage to the lateral prefrontal cortex in monkeys causes a loss of inhibitory control in attentional selection, highlighting the dissociable processes of reversal shift and ED shift learning.

Later research by the Dias et al. (1997) investigated ID and ED shifts in marmosets with lesions in either dorsolateral or orbital (ventromedial) frontal cortex. They found selective impairment of ID reversals with orbital lesions, selective impairment of ED shifts with dorsolateral lesions, and no significant changes on ID shifts with either frontal lesion. The authors interpreted these results as suggesting that distinct regions of the prefrontal cortex perform independent but complementary cognitive processes of visual stimuli in changing environmental circumstances. That is, regions within the orbitofrontal cortex enable the rapid reversal of affective associations for specific visual stimuli, whereas the higher order shifting of attention between features of the visual stimuli is mediated by regions within the lateral prefrontal cortex.

Rogers et al. (2000), using PET, confirmed the dissociability of the ID/ED Task processes. They reported activations in prefrontal regions, including left anterior prefrontal cortex and right dorsolateral prefrontal cortex in ED learning relative to ID shift learning. In contrast, reversal learning, relative to ID shift learning, produced activations of the left caudate nucleus with little change in the dorsolateral prefrontal cortex.

In the specific case of the ID/ED Set Shift Task, it appears that reversal shift and ED shift learning are highly dissociable in both experimental primates and human subjects. For example, lesions of the orbitofrontal cortex in non-human primates have been found to impair reversal learning, but not ED shift learning, with lesions of the dorsolateral prefrontal cortex producing the opposite pattern of deficits (Dias et al., 1996b; Dias, Robbins & Roberts, 1997). Similarly, depletions of the ascending cholinergic projections from the basal forebrain appear to produce highly specific

deficits in reversal learning (Roberts, Robbins, Everitt & Muir, 1992), while depletion of prefrontal dopamine has its greatest effect on ED shifting (Roberts et al., 1994).

These results suggest that distinct regions of the prefrontal cortex carry out independent but complementary forms of cognitive processing of stimuli in changing environmental situations. That is, regions within the orbitofrontal cortex enable the rapid reversal of associations for stimuli, whereas the shifting of attention between features of visual stimuli, such as the perceptual dimensions, is mediated by regions within the lateral prefrontal cortex (Dias et al., 1996a).

The neurophysiological evidence and the effects of lesions described thus suggest that one function mediated by the orbitofrontal cortex is rapid stimulus-reinforcement association learning and the correction of these associations when reinforcement contingencies in the environment change. Findings such as those described above have led to suggestions that the orbitofrontal cortex is primarily involved in the reappraisal of the affective or motivational significance of stimuli and thus plays an important role in emotion-related learning (Rolls, 2004). This emphasises the fact that reappraisal is likely to play an important role in social contexts, where one is required to adapt to rapidly changing contexts (Happaney et al., 2004). This failure to correct behaviour in response to environmental demands may thus underlie the emotional changes and behavioural problems that can follow damage to this region in humans (Rolls et al., 1994).

7.2.3 Response reversal and aggression

Single cell recordings and lesions studies have demonstrated that the orbitofrontal cortex is the substrate of flexible encoding of stimulus reward value in both animals and humans (Rolls, 2000). Parallel to this, damage to the ventral

prefrontal cortex has been observed to result in personality changes, impaired impulse control, and alterations in emotional and motivational state (Mesulam, 2002). It has thus been suggested that some of these changes are related to fundamental alterations in flexible stimulus-reward learning. That is, the socially inappropriate behaviour of frontally-damaged patients may reflect a basic inability to modify ongoing behaviour in response to negative feedback (Rolls et al., 1994).

Decoding the reinforcement value of stimuli enables one to specify goals for action which is vital in motivational and emotional behaviour. With respect to emotional behaviour, decoding and rapidly adjusting the reinforcement value of visual signals is likely to be crucial, due to the fact that emotions are elicited by rewarding and punishing stimuli (Rolls et al., 1994). For example, fear is a state produced by a stimulus associated with a punisher such as pain. The ability to perform this learning rapidly is important in social situations when reinforcements are constantly being exchanged and the reinforcement value of stimuli must continually be updated based on the reinforcement received and given. Thus, any failure to alter behaviour when the reinforcement value of environmental stimuli change will lead to inappropriate emotional and social behaviour.

Some of the personality changes that can follow frontal lobe damage may be related to a dysfunction in altering behaviour in response to a change in reinforcement contingencies (Anderson et al., 1999). Indeed, insofar as the orbitofrontal cortex is involved in the disconnection of stimulus-reinforcement associations, and such associations are important in learned emotional responses, then it follows that the orbitofrontal cortex is involved in emotional responses by correcting stimulus-reinforcer associations when they become inappropriate (Rolls, 2000).

Response reversal deficits may also lead to greater frustration in provocative social interactions. Response reversal involves the ability to make a new response to gain a reward when the old behaviour that previously gave rise to the reward no longer does. In this way, frustration, which is a primary trigger for impulsive-aggression (Berkowitz, 1993), may result if individuals cannot adjust their behaviour and consequently continue to make the old response that no longer results in reward (Blair, 2004).

Patients with ventral frontal lobe damage displaying emotional and social problems have been shown to have deficits on tasks of extinction and reversal of visual discriminations. Rolls et al. (1994) examined the behavioural ratings given by the carers of patients with ventral frontal lobe damage on the Behaviour Questionnaire, including disinhibition or socially inappropriate behaviour, misinterpretation of others' moods, impulsiveness, unconcern or underestimation of the seriousness of their condition, and lack of initiative. Such behavioural changes correlated significantly with the stimulus-reinforcer reversal and extinction learning impairment. Therefore, the difficulty with such correction of behaviour to environmental reinforcers may at least party underlie the behavioural problems of such patients.

Berlin et al. (2004) investigated the relationship between frontal dysfunction, impulsive behaviour, and the behavioural, emotional, and personality changes seen in patients with orbitofrontal cortex damage. These patients performed more impulsively on both self-report and cognitive/behavioural tests of impulsivity, reported more inappropriate „frontal' behaviours, and performed more poorly on a stimulus-reinforcement association reversal task, than non-orbitofrontal cortex lesion controls

and normal controls. Furthermore, orbitofrontal patients experienced more subjective anger than controls.

The relationship between aggression and response reversal and extinction may also be explained in terms of learning theory of premeditated-aggression. That is, premeditated-aggressive individuals may fail to reverse socially unacceptable responses due to positive reinforcement, for example, social power, prestige, or goal attainment. Mitchell et al. (2002) explored performance of adult psychopathic individuals and controls on the ID/ED Task. The psychopathic individuals were significantly impaired in response reversal on this task. On a similar novel response reversal task, Budhani, Richell and Blair (2006) found that adult psychopathic individuals presented with impairment on the response reversal component but not on the acquisition component of the task. On the basis of this it is proposed that premeditated-aggressive individuals are unable to form associations between actions and punishment and thus continue to engage in aggressive behaviours to obtain their desired goal.

7.3 Decision-making

Decision-making requires the evaluation of multiple response options, followed by the selection of the response which is considered to be optimal. Each response option may be characterised by the reward and punishment outcomes with which it is associated. Response options may vary in terms of (1) the degree of reward and punishment; (2) the probability of receiving reward or punishment; and (3) the delay until the reward or punishment is received. This framework provides scope for a range of decision-making abnormalities in clinical groups.

Deficits in decision-making may become apparent in conditions of increased sensitivity to reward or reduced sensitivity to punishment, or at a more complex level, under situations of conflict. For example, a failure to avoid rewards with long-term negative consequences, or the preference for a small immediate reward over a larger delayed reward. This latter phenomenon represents the relationship between decision-making and impulsivity in that an operational definition of impulsive behaviour is the tendency to choose a small immediate reward over a larger delayed reward. In this way, deficits in decision-making may be described as a type of cognitive impulsiveness (Evenden, 1999).

7.3.1 Decision-making and the frontal lobes

Patients with damage involving the orbitofrontal cortex have been reported to display severe impairments in real life decision-making, despite remaining unimpaired intellectually and on traditional neuropsychological measures (Eslinger & Damasio, 1985; Shallice & Burgess, 1991). This syndrome, labelled ‚acquired sociopathy', is characterised by repeated engagement in high risk behaviours that are rewarding in the short term but have likely negative consequences for the individual. The engagement in such behaviours has been proposed to arise from impaired decision-making between various response options (Bechara et al., 2000a).

7.3.2 Decision-making measures

Neuropsychological studies of decision-making in humans have utilised two paradigms in behavioural studies: The Iowa Gambling Task (Bechara et al., 1994), and the Cambridge Gambling Task (Rogers et al., 1999b). The Iowa Gambling Task emphasises the learning of reward and punishment associations in order to guide

ongoing decision-making. Healthy subjects performing the task learn to avoid „risky card decks' that offer high immediate rewards with a concomitant risk of occasional very high punishment. They develop a preference instead for „safe card decks' where the immediate rewards are smaller but there is a low risk of punishment. Patients with bilateral damage to the ventromedial prefrontal cortex do not acquire a preference for the safe decks on the Iowa Gambling Task, but instead prefer the risky decks for the duration of the task (Bechara et al., 1994, Bechara, Damasio, Tranel & Anderson, 1998; Bechara et al. 2000b). In a more recent study, Clark, Manes, Antoun, Sahakian and Robbins (2003) found that the volume of damage in the medial prefrontal cortex and middle and superior frontal gyrus on the right side was significantly correlated with gambling performance. On the basis of these findings, the ventromedial prefrontal cortex has been posited to mediate the learning and retrieval of the affective information that guides decision-making (Clark, Cools & Robbins, 2004). In this way, ventromedial patients' performance on the gambling task is comparable to their real life inability to decide advantageously in situations involving a choice between immediate versus delayed reward or punishment (Bechara et al., 2000b).

The Cambridge Gambling task was developed to quantify decision-making outside of a learning context. The information needed to make each decision is presented to the subject on each trial, and hence the learning demand across trials is minimised. In the Cambridge Gambling Task, both the probabilistic judgement and the betting decision involve the adaptation of behaviour, on a trial by trial basis, according to changes in emotional significance of the stimuli. A failure to adapt over trials may be associated with a reduced ability to moderate the bet according to the associated risk (Rogers et al., 1999b).

To date, four studies have examined performance on the Cambridge Gambling Task in patients with frontal lobe pathology affecting the ventral prefrontal cortex. Increased betting relative to matched controls have been shown in patients with subarachnoid haemorrhage of the anterior communicatory artery, the blood vessel that supplies the ventral prefrontal cortex (Mavaddat, Kirkpatrick, Rogers, & Sahakian, 2000), frontal variant fronto-temporal dementia (Rahman et al., 1999), and large prefrontal lesions including orbitofrontal cortex, caused by haemorrhage or tumour resection (Manes et al., 2002). Whilst probabilistic judgment was at the level of healthy controls in these three studies, the latencies to make those judgments were increased, possibly reflecting an incipient deficit. Surprisingly, however, patients with unilateral lesions restricted to the orbitofrontal cortex performed similarly to controls on the task. In a fourth study, patients with prefrontal lesions including the orbital region showed poorer probabilistic judgment than brain-damaged controls without orbitofrontal damage, and placed lower bets (Rogers et al., 1999a). Probabilistic judgment and betting on the task therefore appear to be closely linked, and this relationship is consistently disrupted by damage to ventral prefrontal cortex.

Neuroimaging studies, primarily using PET, have confirmed activation of the ventromedial prefrontal cortex during the similar Iowa Gambling task performance (Bolla et al., 2003; Ernst et al., 2002). Furthermore, two fMRI studies have examined blood oxygenation level-dependent (BOLD) activity during the Iowa Gambling task (Fukui, Murai, Fukuyama, Hayashi & Hanakawa, 2005; Windmann et al., 2006), with both studies confirming a role for the medial frontal gyrus in the task. Neuroimaging studies have also implicated the ventral prefrontal involvement in the Cambridge Gambling Task (Rogers et al., 1999b; Rubinsztein et al., 2001). Furthermore, Rogers et al. found that contrasts of the decision-making condition with a visuo-motor control

task revealed significant activations in ventral prefrontal cortex including the orbitofrontal gyrus.

The amygdala is a further structure implicated in decision-making circuitry. Iowa Gambling Task deficits are pronounced in patients with amygdala damage, although these patients show a dissociable autonomic profile. Whereas control subjects performing the Iowa Gambling Task developed an ‚anticipatory' SCR prior to selection from the risky decks (Bechara et al., 1996), patients with amygdala lesions did not acquire anticipatory SCRs to punishing feedback (Bechara, Damasio, Damasio & Lee, 1999). Patients with ventromedial prefrontal lesions also failed to acquire anticipatory SCRs to the risky decks, but show comparable SCRs to controls in response to punishing feedback (Bechara et al., 1996). This suggests that at a cognitive level, ventromedial prefrontal patients experience reward and punishment normally, but are unable to use the experiences to guide future behaviour. In comparison, decision-making deficits seen in patients with amygdala damage are due to blunted emotional responses in the initial processing.

7.3.3 Decision-making and aggression

The ability to make decisions, particularly when faced with novel and/or complex problems, is a fundamental skill for functioning in everyday life. Broomhall (2005), in his investigation of neuropsychological deficits in reactive and instrumental violent offenders, found that the reactive violent offenders sampled significantly more cards from the disadvantageous decks on the Iowa Gambling task compared with instrumental violent offenders. The result was taken to indicate reactive violent offenders' real life impairment in the ability to make advantageous choices, particularly in social or personal matters.

In such situations, the exact future outcome of a particular course of action is not possible to determine and choices must be estimations. This suggests that impulsive violent offenders are primarily guided by the immediate prospects and generally insensitive to the possible consequences of their actions. Interestingly, the instrumental group were significantly impaired on the gambling task compared with the normal sample. Broomhall (2005) suggested that the instrumental group may have been able to see the future consequences of their actions, however were still enticed by the high risk decks into making risky choices. This is analogous to their aggressive behaviour in real life situations, in that although there is risk of punishment in acting aggressively, the possibility of gain outweighs this risk.

The data on psychopaths' performance on the gambling task is equivocal (Blair, 2002; Mitchell et al., 2002). Schmitt, Brinkley and Newman (1999) and Blair and Cipolotti (2000) in a small group sample, found that adult psychopathic individuals were comparable to controls in their ability to avoid risky packs. However, both of these studies used task instructions that differed from those of Bechara et al. (1994). Specifically, there were no instructions informing participants that some decks involve more loss than others and that participants could win more money overall if they avoided the costly decks. In the only study where such instructions have been used, the adult psychopathic individuals were significantly less likely than the comparison individuals to avoid the risky packs (Mitchell et al., 2002). This finding can be explained through the identified involvement of the amygdala in stimulus-reinforcement acquisition (Killcross, Robbins & Everitt, 1997). That is, given that amygdala dysfunction is associated with psychopathy (Blair, 2004), it may be through this route rather than the orbitofrontal system that psychopaths have impairment in instrumental learning.

7.3.4 Somatic marker hypothesis

The decision-making research described above has been guided by the „somatic marker hypothesis'. The central idea of this hypothesis is that decision-making is a process guided by emotion in that there is a link between the abnormalities in emotion of orbitofrontal patients and their impairment in decision-making in real life (Bechara et al., 2000a).

This hypothesis proposes that the emotions evoked by the experience of reward or punishment signal the potential occurrence of an outcome, so that these signals guide the behaviour in a manner that is advantageous to the individual. Individuals make judgements not only by assessing the severity of outcomes and their probability of occurrence, but also, in terms of their emotional quality. Lesions of the ventromedial prefrontal cortex interfere with the normal processing of emotional signals leading to pathological impairments in everyday decision-making (Bechara et al., 2000a).

The somatic marker hypothesis and the experimental strategies used to study decision-making in neurological patients provide parallels and direct implications for understanding the nature of clinical disorders. For example, substance abusers are similar to ventromedial patients in that when faced with a choice that brings some immediate reward (i.e., taking a drug), at the risk of incurring a loss of job, home, and family, they choose the immediate reward and ignore the future consequences. Using gambling tasks, studies have indicated that impairment in decision-making may stand at the core of the problem of substance abusers (Bechara & Damasio, 2002; Grant, Contoreggi & London, 2000). This hypothesis may extend to both impulsive- and premeditated-aggressors. That is, such individuals are unable to consider the negative

consequences associated with behaving aggressively, instead focusing on the immediate reaction to the altercation.

7.4 Impulsivity, response reversal, decision-making, and aggression

Multiple parallels exist between the effects on reversal learning and decision-making, which supports the hypothesis that these paradigms measure closely related constructs. As outlined above, Rahman et al. (1999) showed that fronto-temporal dementia is characterised by increased betting on the Cambridge Gambling Task, and impaired reversal on the ID/ED Task, in the presence of intact performance on ED shifting and other dorsolateral prefrontal measures. Furthermore, symptomatology in fronto-temporal dementia resembles the disinhibition syndrome seen after lesions to the orbitofrontal cortex (Gregory & Hodges, 1996).

A combined impairment in decision-making and reversal learning was also observed in a study on psychopathic individuals, selecting more cards from the risky decks on the Iowa Gambling Task, and making more reversal errors on the ID/ED Task (Mitchell et al., 2002). Thus, psychopathic individuals did not adjust their behaviour to avoid making risky decisions and they did not shift their responding when the previously rewarded stimulus was no longer rewarded. A second report in boys with psychopathic tendencies identified a further dissociation, with these subjects making more risky selections on the Iowa Gambling Task but showing intact reversal learning on the ID/ED Task (Blair, Colledge & Mitchell, 2001).

It is important to note here that the aggression shown by psychopathic individuals is predominantly instrumental; it is directed toward a specific goal (Cornell et al., 1996). In comparison, patients with orbitofrontal cortex lesions show primarily impulsive-aggression (Anderson et al., 1999). Thus, it may be that

individuals displaying impulsive- or premeditated-aggression may have dysfunction in similar neuro-circuitry mediating response reversal and decision-making abilities.

7.5 Aim and hypotheses

In summary, lesions of the ventromedial prefrontal cortex, including orbitofrontal cortex, impair reversal learning, decision-making and inhibition in both humans and animals. Functional neuroimaging studies provide convergent evidence, demonstrating ventral prefrontal cortex responses during performance of decision-making and reversal learning in healthy subjects. Disruption in one or all of these abilities may produce a profound alteration in day-to-day functioning.

The aim of this study was to investigate (1) inhibition, as assessed by the Stop Signal Task; (2) response reversal, as assessed by the ID/ED Set Shift Task; and (3) decision-making, as assessed by the Cambridge Gamble Task, in impulsive- and premeditated-aggressive individuals relative to controls. Given the proposed correlation between impulsivity, aggression, and executive function deficits, it was hypothesised that the impulsive-aggressive group would make more errors and have shorter response times on the Stop Signal Task than the premeditated-aggressive and control groups.

Both patients with lesions to the orbitofrontal cortex displaying high levels of impulsive-aggression, as well as psychopaths, characterised by high levels of premeditated-aggression show impairment on tasks of response reversal. It was thus hypothesised that on the ID/ED Task, both the impulsive-aggressive and premeditated-aggressive individuals would be impaired in stimulus-reinforcement and reversal learning, suggesting that such individuals are unable to modify their

behaviour appropriately to changes in reinforcement in a dynamic social environment (Blair, 2004).

Furthermore, it was hypothesised that the impulsive-aggressive group would be impaired in decision-making on the Cambridge Gamble Task. This is based on the premise that such individuals are impaired in their decision-making capacity which result in aggressive responding during social interactions. In contrast, premeditated-aggressive individuals, given their controlled and purposeful aggressive responding, would not be impaired in this ability.

7.6 Method

7.6.1 Participants

The participants for Study 3 are the same as those for Study 2.

7.6.2 Materials

7.6.2.1 Questionnaires

The BPAQ-SF (Bryant & Smith, 2001) was used for the purpose of selecting participants who were characteristically aggressive. The IPAS (Stanford et al., 2003a) was employed to characterise the aggressive acts as predominantly impulsive or predominantly premeditated in nature. Participants also completed the BPAQ (Buss & Perry, 1992) and I7 Impulsivity Questionnaire (Eysenck et al., 1985) upon completion of the computer tasks. All questionnaires are outlined in detail in Chapter 5.

7.6.2.2 Inhibition, response reversal, and decision-making tasks

Three subtests from the Cambridge Neuropsychological Test Automated Battery (CANTAB) were chosen as measures of inhibition, response reversal, and decision-making: the Stop Signal Task, ID/ED Set Shift Task and Cambridge

Gambling Task. The CANTAB neuropsychological battery has been extensively validated in brain injury and neuroimaging studies (Fray, Robbins & Sahakian, 1996).

Stop Signal Task: The Stop Signal Task is based on the classic stop signal response paradigm which measures a participant's ability to inhibit a response. Such tasks require subjects to perform speeded responses on „Go' trials and to inhibit their responses on „Stop' trials. This paradigm reflects Barkley's (1999) process of suppressing prepotent responses and stopping ongoing responses. Inhibition, as conceptualised in this study, refers to the deliberate, controlled suppression of prepotent responses. The Stop Signal Task is used to tap this type of inhibition, in that it requires the deliberate stopping of a response that is relatively automatic (Miyake et al., 2000).

Intra/Extradimensional Set Shift Task: The ID/ED Set Shift Task is a measure of rule acquisition and reversal. It features visual discrimination and attentional set formation, as well as maintenance, shifting and flexibility of attention. It requires participants to learn a series of visual discriminations, using feedback provided by the computer, in which one of two stimulus dimensions is relevant and the other is not. An ID shift occurs when a participant trained to respond to a particular stimulus dimension (e.g., shape) is required to transfer the rule to a new set of examples of the same dimension. An ED shift occurs when a participant is required to shift response set to an alternative previously irrelevant dimension (Owen et al., 1991). Two artificial dimensions are used in this test: colour-filled shapes and white lines. Simple stimuli are made up of just one of these dimensions, whereas compound stimuli are made up of both, namely white lines overlying colour-filled shapes.

Cambridge Gambling Task: The Cambridge Gambling Task assesses decision-making and risk-taking behaviour outside a learning context. Relevant information is

presented to participants and it is not necessary for them to learn or retrieve information over consecutive trials. The Cambridge Gambling Task has two modes: ascending first and descending first. In the ascending first mode, the value of the stakes displayed in the stake box rises in stages 2 and 3, and falls in stages 4 and 5. In the descending first mode, the value of the stakes falls in stages 2 and 3 and rises in stages 4 and 5.

The CANTAB tasks were presented on a Paceblade Slimbook P110 touch-screen computer, with a 12 inch monitor. For the Stop Signal Task, participants used a press pad.

7.6.2.3 Wechsler Adult Intelligence Scale – Third Edition (Wechsler, 1997)

As outlined in Chapter 5, the Vocabulary and Digit Span subtests from the WAIS-III were selected as control measures.

7.6.3 Procedure

The tasks for Study 3 were completed at the same time as those for Study 2. As outlined in Chapter 6, participants were recruited from undergraduate psychology classes at the University of Tasmania using the BPAQ-SF. Following the screening process, select participants who qualified as aggressive or control participants were invited to participate in computer tasks for Study 2 and Study 3. All participants received course credit for their participation. Informed written consent was obtained from all individuals prior to participation (see Appendix F and G for participant information sheets and consent forms). Participants were tested individually in a quiet room in the School of Psychology at the University of Tasmania. Presentation of the

tasks for Study 2 and Study 3 were counterbalanced as well as the presentation of the tasks within Study 3.

Participants were seated in front of a computer monitor, approximately 60cm from the screen. Each task was explained and comprehension ensured before beginning.

Stop Signal Task: The task screen for the Stop Signal Task began with a white ring presented on a black background, displayed to focus the attention of the participant. After a 500ms delay, a visual stimulus was displayed, consisting of a white arrow pointing to the left or to the right. The test consisted of two parts. In the first part, the participant was introduced to the press pad, and told to press the left hand button when they saw a left-pointing arrow and the right hand button when they saw a right-pointing arrow. Participants were instructed to use their index fingers to press the key pad. The participant completed one block of 16 trials to practice this. For the second part, the participants were told to continue pressing the buttons on the press pad when they saw the arrows as before, however if they heard an auditory signal (a beep), they should withhold their response and not press the button. There were five assessed blocks, each of 64 trials. Each block was divided into four sub-blocks of 16 trials for analysis purposes only (not evident to the participant). Every sub-block contained twelve ‚go' trials, with no auditory stop signal, and four ‚stop' trials, with an auditory tone played following the ‚stop signal delay' (SSD) period, which is measured from the onset of the arrow stimulus. The twelve ‚go' trials and four ‚stop' trials were given in a random order within each sub-block, but all trials from one sub-block took place before the next sub-block began.

The timing of the auditory stop signal changed throughout the test, depending on the participant's performance, so that stopping occurred approximately 50% of the

time for each participant. The shorter the SSD, the more likely it was that the participant would be able to hold off responding to the arrow. Note, for some participants, the SSD may have become negative, that is, the auditory signal occurred before the onset of the arrow stimulus. At the end of every assessed block, a feedback screen was displayed, displaying a graphical representation of the participant's performance, which the examiner explained to the participant, as well as encouraging them to respond faster. The participant was also shown if their speed of response was improving or not.

Intra/Extradimensional Set Shift task: The ID/ED Task consisted of nine blocks. Block 1 began with the presentation of two simple, colour-filled shapes. The participant had to learn which of the stimuli was correct by touching it, and continue until the criterion was reached. In Block 2, the contingencies were reversed, so that now the previously incorrect stimulus was correct. In Block 3, a second dimension was introduced, initially lying adjacent to, and then, for Block 4, overlapping, the first dimension. The contingencies did not change, remaining the same as at the end of the simple discrimination. The contingencies were reversed for Block 5, within the original dimension. New compound stimuli were introduced in Block 6, still varying along the same two dimensions (of shape and of line). Participants were required to attend to the previously relevant dimension of shape and learn which of the two new exemplars was correct (the „ID shift'). Once the participant successfully completed the ID shift, followed by a reversal (Block 7), the compound stimuli were again changed. For Block 8, participants were required to shift attention to the previously irrelevant dimension and learn which of the two exemplars in this new dimension was now correct (the „ED shift'). In Block 9, the contingencies were again reversed. Participants indicated their responses by touching the identified pattern on the screen.

For each of the nine stages, participants proceeded onto the next stage when a criterion of six consecutive correct responses had been attained. If this criterion was not reached after 50 trials, the computer automatically terminated the test. Performance was examined by the mean number of stages completed and the number of errors made at each stage.

Cambridge Gambling Task: On each trial of the Cambridge Gambling Task, participants were presented with a row of ten boxes across the top of the screen, some of which were red and some of which were blue. At the bottom of the screen were two rectangles, containing the words „Red' and „Blue'. The participant was instructed to guess whether a yellow token was hidden under a red box or a blue box. In the gambling stages, participants started with a number of points which was displayed on the screen. They were to select a proportion of these points (5%, 25%, 50%, 75% or 95%), displayed in either rising or falling order, to gamble on their confidence in the location of the yellow token. A stake box on the screen displayed the current amount of the bet. The task was made up of five stages, with instructions provided at the start of each stage. The first stage was a decision stage only, in which the participant chose whether a token was hidden under a red box or a blue box by touching the appropriate box at the bottom of the screen. The second stage was a training stage for gambling, with either ascending or descending stakes (depending on the test mode), in which the participant first chose whether the token was hidden under a red box or a blue box by touching the appropriate box at the bottom of the screen, and then selecting the amount they wished to bet by touching the stake box on the screen at the appropriate time as the bets were displayed in either ascending or descending order. If the screen was not touched to choose a stake, the final value displayed in the stake box was used. The third stage was the test stage for gambling, in which the participant's

performance was assessed. The fourth stage was a further training stage for gambling, however in this stage, the stakes were offered in the reversed order to the second and third stages (i.e. either ascending or descending). The final stage was the final gambling stage with the stakes offered in the same direction as the fourth stage. The participant's performance was assessed. The order of the ascending and descending presentations was counterbalanced.

7.7 Results

7.7.1 Stop Signal Task

The Stop Signal Task has five outcome measures: (1) Direction Errors measures each trial in which the participant pressed the wrong button (the left button when the right arrow was shown on the screen, or vice versa); (2) Proportion of Successful Stops measures the number of times the participant stopped successfully, divided by the total number of stop signals; (3) Reaction Time on „Go' Trials; (4) SSD refers to the delay at which the participant was able to stop 50% of the time. It is calculated as the arithmetic mean of the measured SSD, or failed-stop reaction time if applicable, from completed assessment stop trials; (5) Stop Signal Reaction Time (SSRT) is an estimate of the length of time between the go stimulus and the stop stimulus at which the participant is able to successfully inhibit their response on 50% of the trials. This measure is calculated from two other SST measures – the reaction time on „Go' trials and the SSD (50%) measure.

Mean scores were analysed using separate one-way ANOVAs. The results of the analysis yielded no significant differences between the three participant groups for direction errors, $F(2, 86) = 0.278$, $MSE = 20.62$, $p = .758$, $\eta^2 = .007$, proportion of successful stops, $F(2, 86) = 0.831$, $MSE = .007$, $p = .439$, $\eta^2 = .019$, mean correct reaction time, $F(2, 86) = 0.394$, $MSE = 2367.03$, $p = .676$, $\eta^2 = .009$, SSD, $F(2, 86) =$

1.48, MSE = 25010.07, p = .233, η^2 = .034, or SSRT, F(2, 86) = 1.78, MSE = 13331.41, p = .758, η^2 = .041. See Table 7.1 for means and standard deviations.

Table 7.1

Means (and standard deviations) for the Stop Signal Task for the three participant groups

	Impulsive-Aggressive	Premeditated-Aggressive	Control	Total
Direction Errors	6.86 (8.54)	8.27 (11.19)	5.27 (6.19)	6.16 (8.53)
Proportion of Successful Stops	0.44 (0.11)	0.47 (0.07)	0.47 (0.08)	0.46 (0.09)
Mean Correct RT	387.52 (66.71)	399.36 (75.43)	404.11 (89.92)	396.24 (77.01)
SSD	140.66 (136.62)	188.14 (122.05)	190.55 (127.38)	169.87 (130.65)
SSRT	222.49 (96.66)	191.59 (81.03)	184.10 (77.77)	201.44 (87.45)

7.7.2 Intra/Extra Dimensional Set Shift task

The nine outcome measures for the ID/ED are divided into two groups; errors and numbers of trials and stages completed.

Errors: (1) Pre-ED Errors are the number of errors made prior to the extra-dimensional shift of the task. Errors are defined as instances when the participant fails to select the stimulus that is compatible with the current rule; (2) ED Shift Errors are those errors made in the ED stage of the task as they have been committed at the stage where the participant is required to make an ED shift. Errors committed at the reversal

stage following the ED shift stage are not included; (3) Total Errors is a measure of the participant's efficiency in attempting the test. Thus, whilst a participant may pass all nine stages, a substantial number of errors may be made while doing to. It is important to note that participants failing at any stage of the test have had less opportunity to make errors. The Total Errors (adjusted) measure attempts to compensate for this: (4) Total Errors (adjusted) is a measure of the participant's efficiency in attempting the test. The adjusted score is calculated by adding 25 for each stage not attempted due to failure. This value of 25 is used since most participants must complete 50 trials to fail a stage and half of these could be correct by chance alone; (5) Completed Stage Errors is the number of errors made on stages successfully completed.

Number of Trials and Stages Completed: (1) Stages Completed is the total number of stages the participant completed successfully; (2) Total Trials is the number of trials completed on all attempted stages. Subjects failing at any stage of the task have had less opportunity to complete trials – the Total Trials (adjusted) measure attempts to compensate for this; (3) Total Trials (adjusted) is the number of trials completed on all attempted stages with an adjustment for any stages not reached. The adjustment adds 50 for each stage no attempted due to failure at an earlier stage; (4) Completed Stage Trials is the number of trials undertaken on all successfully completed stages.

Mean scores for the nine outcome measures were analysed with separate univariate ANOVAs (see Table 7.2 for means and standard deviations). The results of the analysis yielded non-significant differences between the three participant groups on all outcome measures ($p>.05$). See Table 7.3 for results of ANOVA.

Table 7.2

Means (and standard deviations) on the Intra-Extra Dimensional Set Shift Task for the three participant groups

	Impulsive-Aggressive	Premeditated-Aggressive	Control	Total
Completed Stage Errors	11.57 (7.28)	9.59 (3.26)	10.93 (5.50)	10.85 (5.86)
Completed Stage Trials	70.51 (16.09)	67.55 (8.06)	69.73 (14.73)	69.49 (13.88)
ED Shift Errors	6.66 (8.43)	6.50 (8.33)	4.30 (5.65)	5.81 (7.55)
Pre-ED Errors	6.14 (2.02)	6.14 (2.38)	6.70 (4.19)	6.33 (2.99)
Stages Completed	8.71 (0.67)	8.64 (0.79)	8.70 (1.32)	8.69 (0.96)
Total Errors	17.89 (13.99)	13.81 (8.42)	12.57 (7.30)	15.02 (1089)
Total Errors (adjusted)	19.03 (16.34)	18.36 (17.95)	18.40 (31.24)	18.64 (22.66)
Total Trials	79.09 (18.57)	76.64 (15.78)	73.07 (13.02)	76.39 (16.16)
Total Trials (adjusted)	84.80 (28.92)	85.73 (34.82)	84.73 (56.20)	85.01 (41.13)

7.7.3 Cambridge Gambling Task

The Cambridge Gambling Task has six outcome measures: (1) Quality of Decision-Making is the proportion of trials on which the participant chose to gamble on the more likely outcome; (2) Deliberation Time is measured from the presentation of the coloured boxes to the participant's choice of colour; (3) Risk Taking reports the mean proportion of the current points total that the participant chose to risk on gamble test trials for which they had chosen the more likely outcome (i.e., trials on which

Table 7.3

Results of univariate ANOVAs for the Intra/Extra Dimensional Set Shift task

Variable	df	F	MSE	*p*	η^2
Errors					
Pre-ED Errors	2, 86	0.34	3.08	.714	.008
EDS Errors	2, 86	0.91	52.00	.406	.021
Total Errors	2, 86	2.17	249.89	.121	.049
Total Errors (adjusted)	2, 86	0.01	4.35	.992	.000
Completed Stage Errors	2, 86	0.77	26.65	.465	.018
Number of Trials and Stages Completed					
Stages Completed	2, 86	.05	0.04	.955	.001
Total Trials	2, 86	1.13	293.51	.329	.026
Total Trials (adjusted)	2, 86	0.004	7.58	.996	.000
Completed Stage Trials	2, 86	.31	60.84	.734	.007

they had more chance of winning than losing); (4) Risk Adjustment reflects the tendency of participants to bet a higher proportion of their points on trials when the large majority of boxes are the colour chosen than when a smaller majority of the boxes are of the colour chosen; (5) Delay Aversion measures the tendency for participants who are unable or unwilling to wait to bet larger amounts when the possible bet amounts are presented in descending order than they do when the amounts are presented in ascending order. It is calculated by subtracting the Risk Taking measure, calculated for ascending gamble trials, from the Risk Taking

measure, calculated for descending trials; (6) Overall Proportion Bet reports the average proportion of the current points total that the participant chose to risk on each gamble test trial, including trials on which they bet on the less likely outcome, and trials on which both outcomes were equally likely.

Mean scores for each of the six outcome measures were analysed using separate univariate ANOVAs (see Table 7.4 for means and standard deviations). The results of the analyses yielded a non-significant effect of group for all outcome measures (*p*s.>.05) with the exception of Risk Adjustment (descending order). Post hoc Tukey's indicated that in comparison to the premeditated-aggressive group, the control group bet a higher proportion of their points on trials when the large majority of boxes were the colour chosen, than when a smaller majority of the boxes were the colour chosen (*p*s.<.05). See Table 7.5 for ANOVA results.

Table 7.4

Means (and standard deviations) on the Cambridge Gambling Task for the three participant groups

	Impulsive-Aggressive	Premeditated-Aggressive	Control	Total
Quality of Decision Making				
Total	0.96 (0.08)	0.94 (0.10)	0.97 (0.07)	0.96 (0.08)
Ascending	0.96 (0.08)	0.94 (0.12)	0.96 (0.08)	0.95 (0.09)
Descending	0.96 (0.10)	0.95 (0.11)	0.98 (0.07)	0.96 (0.09)
Deliberation Time				
Total	1441.29 (328.86)	1453.49 (490.81)	1648.02 (451.77)	1515.66 (423.97)
Ascending	1458.21 (452.52)	1468.93 (610.95)	1701.89 (494.93)	1544.95 (517.53)
Descending	1421.43 (408.74)	1454.14 (476.30)	1559.97 (618.33)	1477.47 (504.15)
Risk Taking				
Total	0.57 (0.14)	0.53 (0.16)	0.56 (0.11)	0.56 (0.14)
Ascending	0.42 (0.19)	0.41 (0.16)	0.43 (0.13)	0.42 (0.16)
Descending	0.73 (0.15)	0.71 (0.20)	0.70 (0.14)	0.71 (0.16)
Risk Adjustment				
Total	1.14 (0.79)	1.48 (0.94)	1.65 (1.02)	1.40 (0.93)
Ascending	1.89 (0.83)	2.25 (1.12)	1.98 (1.12)	2.01 (1.01)
Descending	0.85 (0.89)	1.18 (0.92)	1.49 (1.06)	1.15 (0.99)
Delay Aversion	0.31 (0.22)	0.31 (0.22)	0.27 (0.15)	0.30 (0.20)
Overall Proportion Bet				
Total	0.54 (0.12)	0.51 (0.14)	0.52 (0.11)	0.53 (0.12)
Ascending	0.38 (0.17)	0.36 (0.15)	0.39 (0.12)	0.38 (0.15)
Descending	0.70 (0.16)	0.67 (0.20)	0.66 (0.15)	0.68 (0.16)

Table 7.5

Results of univariate ANOVAs for the Cambridge Gambling Task

Variable	df	F	MSE	*p*	η^2
Quality of Decision Making					
Total	2, 86	0.63	0.005	0.534	.015
Ascending	2, 86	0.69	0.006	0.503	.016
Descending	2, 86	0.61	0.005	0.545	.014
Deliberation Time					
Total	2, 86	2.31	402109.86	0.106	.052
Ascending	2, 86	2.17	564685.48	0.121	.049
Descending	2, 86	0.64	163026.79	0.532	.015
Risk Taking					
Total	2, 86	0.64	0.012	0.53	.015
Ascending	2, 86	0.11	0.003	0.894	.003
Descending	2, 86	.042	0.013	0.626	.011
Risk Adjustment					
Total	2, 86	2.59	2.15	0.081	.058
Ascending	2, 86	0.87	0.89	0.422	.020
Descending	2, 86	3.60	3.32	0.032*	.079
Delay Aversion	2, 86	0.45	0.02	0.642	.010
Overall Proportion Bet					
Total	2, 86	0.31	0.004	0.737	.007
Ascending	2, 86	0.30	0.007	0.739	.007
Descending	2, 86	0.74	0.02	0.482	.017

7.8 Discussion

In this study, subtests from the CANTAB neuropsychological battery were used to investigate possible inhibition, response reversal and decision-making

dysfunction in impulsive- and premeditated-aggressive individuals. Although this is the first report on the use of this battery with an aggressive sample, the findings can be compared with a number of other studies using CANTAB to assess neuropsychological function in a variety of disorders including APD (Dolan & Park, 2002), brain injury (Owen et al., 1991; 1993), schizophrenia (Elliott, McKenna, Robbins & Sahakian, 1995; Pantelis et al., 1997), depression (Elliott et al., 1996; Sweeney, Kmiec & Kupfer, 2000) and ADHD (Kempton et al., 1999). Contrary to prediction, there were no significant differences in performance between the impulsive-aggressive, premeditated-aggressive or controls on tests of inhibition, response reversal, or decision-making, all of which to a greater or lesser extent depend on the functional integrity of the orbitofrontal cortex.

7.8.1 Stop Signal Task

On the Stop Signal Task, it was anticipated that impulsive-aggressive individuals would exhibit performance deficits (e.g., shorter response times and higher number of errors) than the premeditated-aggressive and control groups. This hypothesis was based on previous findings suggesting a relationship between the propensity for impulsive-aggression and executive and inhibition deficits. The results from the Stop Signal Task do not support this hypothesis, finding no differences between the three participant groups on this measure.

Several studies have demonstrated that performance deficits on the Stop Signal Task are associated with medial and inferior frontal lobe functioning (see Aron et al., 2007b; Chambers et al., 2009; Verbruggen & Logan, 2008 for reviews). This suggests that the inhibitory deficits mediated by such regions of the prefrontal cortex are not responsible for the display of impulsive-aggression in a subclinical population.

In contrast however, Dolan and Park (2002) found that APD subjects had significantly greater difficulty in inhibiting a prepotent response than controls on the similar Go/No-Go task. Such discrepant findings suggest distinct neural mechanisms underlying specifically characteristic aggression in comparison to antisocial behaviour more broadly.

The lack of significant group differences on this measure may be relative to the fact that aggression contains a social component and the Stop Signal Task does not. There is evidence to suggest that prefrontal cortex is involved in the processing of both motivational and emotional stimuli and moreover that prefrontal lesions may lead to disruptions in social behaviour (Anderson et al., 1999; Damasio, 1995). Such disruption may occur, however, only in situations of heightened arousal such as hostile social interactions. In this way, the increased levels of frustration and anger impede on adequate social-information processing leading to a retaliatory response. This is in stark contrast to the environment in which the Stop Signal Task was performed when participants were not experiencing anger and could successfully attend to the task at hand.

Thus, although this study found no evidence of an inhibitory deficit in the impulsive-aggressive group, this result may be specific to non-social situations. Aggression is a primal social response option for social situations. This may become the chosen reaction in an interpersonal interaction when the individual is faced with complex contextual cues. As Robbins (1998) described, executive function involves the ability to formulate new plans of action, to select appropriate responses, and to monitor behaviour with respect to one's emotional state. It may then be that impulsive-aggressive individuals, who have executive functioning deficits, demonstrate poor social information processing skills and are unable to cope with

overwhelming response options. As a consequence, they fail to access more socially appropriate response options and make default aggressive responses in provocative situations despite experiencing regret for reacting in this manner.

The use of ERPs can help to elucidate the relationship between underlying the impulsivity personality trait and inhibition. Comparable to the present study, Dimoska and Johnstone (2007) found non-significant group differences on the Stop Signal Task in non-clinical adults who scored in the top and bottom 15% on the I7 Impulsivity Questionnaire (Eysenck et al., 1985). Interestingly, however, the results revealed that lateralised readiness potential amplitude on „Stop' trials was larger in the High impulsivity than Low impulsivity group, indicating greater response side-specific motor preparation in the primary motor cortex. An N1/P3 complex was also evoked on successful „Stop' trials and was enhanced in the High group in comparison to the Low group. The successful-stop P3 has previously been implicated as an index of inhibition in the primary motor cortex, suggesting enhanced inhibitory processing in the High group. These findings indicate that the High impulsivity group was able to counteract an impulsive response style on stop-signal trials with enhanced inhibitory activation, resulting in comparable overt performance to the Low impulsivity group.

In the Stop Signal Task, the P3 has been linked to the response inhibition process (Kok, Ramautar, de Ruiter, Band & Ridderinkhof, 2004), with a larger successful-stop P3 component in fast compared to slow responders suggesting that the component may reflect inhibition activated when an urgent inhibitory responses is required (Dimoska, Johnstone & Barry, 2006). A reduced or delayed P3 on inhibition trials, indicating an impaired response inhibition process, has been reported in a number of clinical populations characterised by impulsivity including children (Overtoom et al., 2002) and adults with ADHD (Bekker et al., 2005). As outlined

above, however, Dimoska & Johnstone (2007) found that in non-clinical subjects who reported high degrees of impulsiveness showed enhanced response inhibition as evidenced by a larger N1/P3 complex. These results suggest that non-clinical impulsive individuals are able to compensate an impulsive response style on stop-signal trials with enhanced inhibitory activation, resulting in comparable overt performance to those reporting low impulsiveness. The authors argued that such findings indicate that a high degree of trait impulsivity in non-clinical adults is not due to a deficiency in the response inhibition process.

The personality trait impulsivity is typically measured using self-report measures. In the laboratory, however, impulsivity has been operationalised as an inability to inhibit a behavioural or cognitive response, and is measured through tasks such as the Stop Signal Task. To date, however, it remains unclear whether a deficient response inhibition mechanism may underlie the personality trait impulsivity in non-clinical populations. Correlational studies in non-clinical populations have reported that higher scores on impulsiveness questionnaires correspond with longer stop signal reaction times (Gorlyn, Keilp, Tryon & Mann, 2005), reduced inhibition rates (Keilp, Sackeim & Mann, 2005), or general stopping problems (Vigil-Colet & Codorniu-Raga, 2004). However, a number of studies, including the present investigation, have failed to find a reliable relationship between impulsiveness and stop signal reaction times. When examining extreme high and low non-clinical impulsivity groups, several studies reported no differences between groups (Dimoska & Johnstone, 2007; Lijffijt et al., 2004; Rodriguez-Fornells, Lorenzo-Seva & Andres-Pueyo, 2002). Furthermore, factor analyses suggest self-report measures of the impulsiveness trait and laboratory measures of response inhibition and impulsivity may reflect distinct constructs (Dolan & Fullam, 2004; Reynolds, de Wit & Richards, 2002). Thus, although self-reporting

high levels of impulsivity, behavioural measures of this trait do not indicate inhibitory deficits in the response inhibition process.

Inhibition is important in the development of emotional self-regulation, that is, those who have deficiencies in inhibition should demonstrate greater emotional reactivity to emotionally charged events as well as less capacity to regulate emotional states in the action of goal-directed behaviour (Barkley, 1997). Disorders of emotion are common in individuals with injury sustained to the prefrontal cortex, which suggests that this region is critical not only for inhibition but also for the self-control of emotion (Davidson et al., 2000b). Given the findings of the current study, it may be suggested that performance on the Stop Signal Task does not reflect inhibitory dysfunction present in impulsive-aggressive individuals. While the Stop Signal Task is a valid measure of inhibition, it may be the combination of heightened arousal, anger, and information processing deficits coupled with an impulsive personality that give rise to the display of impulsive-aggression. Inhibitory deficits in isolation, however, will not cause impulsive-aggression.

7.8.2 Intra/Extradimensional Set Shift task

On the ID/ED Set Shift task, there was no significant difference between the groups on any of the measures, including the number of trials until the first reversal, and the number of trials completed to acquire a positive stimulus. Further, the acquisition task was completed successfully by all participants. This indicates that all participants understood the task demands, could follow task instructions, and learn appropriate stimulus-reinforcement associations. Therefore, in contrast with expectations, impulsive-aggressive and premeditated-aggressive individuals do not

present with reversal impairments, that is, a failure to change behaviours in response to changed reinforcers.

Patients with lesions to the orbitofrontal cortex have been shown to be impaired in forming new stimulus-reward associations when reinforcement contingencies are reversed (Berlin et al., 2004). Furthermore, difficulties adapting behaviour to changes in reinforcement (i.e., response reversal) have been linked with aberrant social behaviour (Rolls, 2004). Blair (2004) argued that the increased rates of impulsive-aggression seen in psychopaths may be due to heightened levels of frustration resulting from a failure to modify their behaviour appropriately to changes in reinforcement in dynamic social environments. Following from this, it was proposed that subclinical aggression, either impulsive or premeditated in nature, would similarly result from such frustration in interpersonal situations when individuals are unable to adjust behaviours. However, the current findings do not indicate such impairment in the present population.

The lack of significant group differences on ED shifts are in contrast to the findings by Dolan and Park (2002) in their sample of APD individuals and Owen et al. (1990, 1991) in patients with frontal lobe damage and with neuro-degenerative disorders involving frontal circuitry. With regard to Dolan and Park, they found that their sample were impaired on the ED shift stage, but not at the ID shift stage of the ID/ED Task, taking the result to implicate the dorsolateral prefrontal cortex in APD through its role in set-shifting ability. In contrast, deficits were not seen on either the ID or ED shift stages of the task in both impulsive- and premeditated-aggressive individuals in the current study, indicating non-impaired functionality of these abilities in this sample. These findings again indicate distinct neuropsychological deficits associated with specifically impulsive- and premeditated-aggression and those

associated with the broader diagnosis of APD. The increased severity of behaviours demonstrated by APD individuals could thus be a result of the further impairment in response reversal.

In line with this argument, Mitchell et al. (2002) found that psychopathic individuals showed a selective response reversal deficit, and similarly, Lapierre et al. (1995) suggested impaired response reversal performance in psychopathic adults. Indeed, psychopaths present with marked psychopathology, including aggressive narcissism and a socially deviant lifestyle (Hare, 1991). In contrast, the present sample, while reporting heightened levels of trait aggression, continue to function adaptively within the community.

It can then be suggested that the degree of dysfunction in the orbitofrontal circuit will determine the degree of response reversal impairment on tasks such as the ID/ED. That is, the impairment evident in the current experimental groups may be less that that in clinical populations used in previous studies (e.g., Mitchell et al., 2002). This difference in the degree of orbitofrontal impairment between these samples could significantly impact upon the severity of the aggressive behaviour displayed.

With regard to premeditated-aggression, those presenting with high levels of premeditated-aggression may not be impaired in reversing responses in negative social interactions as they do not deem aggressive exchanges as a form of punishment. Instead, aggression is viewed as a viable way to attain their desired goal, and in this way, is viewed as a reward for the current behaviour. In line with this, Mitchell et al. (2002) reported that the psychopathic individuals performed similar to comparison individuals on the learning component of the ID/ED Task.

7.8.3 Cambridge Gambling Task

The performance of the impulsive- and premeditated-aggressive groups on the Cambridge Gambling Task did not differ significantly from that of controls. Specifically, all participant groups were able to make appropriate choices related to the uncertain outcomes involved in the decision-making task.

Performance deficits on the gambling task are associated with ventromedial dysfunction in humans (Rogers et al., 1999b; Rubinsztein et al., 2001). Following prior work in the neuropsychiatric literature, it is assumed that the orbitofrontal cortex mediates sensitivity to changing reinforcement contingencies, and thus may be particularly important for modulating individuals' response in social situations. Impaired performance on the Gambling Task may reflect an inability to effectively process feedback information regarding reward and punishment, and thus employ such cues to guide behaviour.

This non-impaired performance of the impulsive-aggressive group on the Gambling Task finding is in contrast to that of Broomhall (2005) in his investigation of neuropsychological deficits in reactive and instrumental violent offenders as well as Mitchell et al. (2002) in psychopathic individuals. These samples were less likely to avoid making risky selections over the course of the gambling task relative to comparison participants. Similarly, Best et al. (2002) reported the performance of psychiatric patients with Intermittent Explosive Disorder. They found that such individuals continued to pick cards from the disadvantageous decks whereas the control subjects learnt to avoid such decks, instead choosing from the advantageous decks. However, while their performance was impaired compared to controls, it was not as impaired as that of neurological patients in previous studies (Bechara, Damasio, Tranel & Damasio, 1997; Bechara et al., 1998; Bechara et al., 1999), indicating that

the impairment associated with Intermittent Explosive Disorder may be milder than after orbital/medial damage.

In everyday life, individuals with Intermittent Explosive Disorder continually use problem-solving strategies that involve aggression, despite it being socially inappropriate and frequently leading to punishment in the form of injury or incarceration. In this way, such individuals show an inability to learn from social cues provided in the environment. Thus, it may be that the impulsive-aggressive individuals in the present study do not have dysfunction within this region of the prefrontal cortex, while those presenting with more severe impulsive-aggression do have such deficits. This is in conjunction with the orbitofrontal circuit hypothesis outlined above. That is, the degree of dysfunction in this cortical circuit determines the degree of severity of aggressive behaviours.

The finding that impulsive-aggressive individuals did not place earlier bets in either the ascending or descending conditions of the task compared with controls suggests that such individuals are able to withhold impulsive responding. This is in contrast to the presumed impairment in withholding responses in order to gain larger immediate rewards which characterises impulsive-aggressive responding. Berlin et al. (2004) suggested that part of the reason orbitofrontal patients are impulsive may be related to a tendency to respond rapidly to rewards and punishers without sufficiently assessing the consequences. That is, orbitofrontal patients may act without giving themselves enough time to think about their behaviours and to modify them accordingly. In contrast, the performance of the impulsive-aggressive individuals on the Cambridge Gambling Task does not suggest impulsive responding in experimental conditions. Such controlled action however may become impaired when combined with heightened levels of anger present in hostile interactions.

With regard to premeditated-aggression, while they are able to make decisions adequately based on information provided to them in social interactions, they may not have experienced punishment following a display of aggression and therefore do not perceive the need to modify their behaviour. Evidence from social interaction theory (Tedeschi & Felson, 1994) suggests that premeditated-aggression may involve decisions on how to achieve goals by making explicit cost-benefit analyses that take into account the probabilities of various outcomes. Premeditated-aggressive individuals often have explicit beliefs that aggression will be a successful means of attaining a particular outcome (Huesmann & Guerra, 1997), or they are inhibited from aggression because of explicit representations of the possible costs (Buss, 2005). Premeditated-aggression is thus facilitated by explicit attitudes on the appropriateness of aggression in certain situations.

This suggestion fits with models emphasising the importance of explicit processing in aggression. An internal state of anger is produced via an aversive experience that results in an automatic appraisal. According to Anderson and Bushman (2002), whether these internal states results in aggression is determined, in the absence of impulsivity, by the outcome of conscious appraisal and decision processes enabled by an effortful control system.

7.8.4 Limitations and directions for future research

The CANTAB neuropsychological battery has been extensively validated in brain injury and neuroimaging studies (Fray et al., 1997). However, to date, relatively little research has been conducted utilising this battery in subclinical populations, and more specifically aggressive populations. One study conducted by Robbins et al. (1998) investigating executive functioning in normal volunteers found that on the ID/ED Set Shift Task, participants under the age of 55 made significantly fewer errors

than the older age groups. Given that the mean errors rate on the more difficult extradimensional shift reversal stage was approximately two in the younger age group, a possible ceiling effect in younger adults on this task may be suggested. Further research is thus required to determine its sensitivity in this population before more definite theories can be postulated regarding the relationship between subclinical aggression and performance on measures of orbitofrontal functioning as purported to be measured by the tasks used in the current study.

To more finely assess the extent to which aggression is related to difficulties inhibiting such responses, adaptation of currently existing cognitive measures of inhibition and impulsivity such as those utilised in the current study should be adapted to use socially relevant stimuli such as facial expressions of affect. Furthermore, neuroimaging techniques such as fMRI and ERPs could be used with subclinical aggressive individuals to assess abnormal activation in regions of the prefrontal cortex, abnormalities which may not be detected in neuropsychological tests.

7.8.5 Conclusion

These data indicate that both impulsive- and premeditated-aggressive individuals do not exhibit impairments on tests of inhibition, response reversal, or decision-making, shown to be mediated by orbitofrontal/ventromedial regions of the prefrontal cortex. While APD individuals and psychopaths have been shown to have deficits on such measures, the lack of significant findings in this study suggests distinct neural mechanisms underlying aggression specifically and antisocial behaviour more broadly. Indeed, psychopathological disorders such as APD and psychopathy may or may not involve an aggressive component. It may thus be suggested that the additional ventromedial deficits in inhibition, response reversal,

and decision-making may contribute to the display of more severe antisocial behaviours as those seen in APD and psychopathy.

Chapter 8

General Discussion

The central aim of the present thesis was to clarify the role of the prefrontal cortex in impulsive- and premeditated-aggression. More specifically, it sought to investigate the separable roles of the dorsolateral and orbitofrontal regions of the prefrontal cortex and how they may mediate the expression of these subtypes of aggressive behaviour.

The first study explored possible executive functioning impairment, pertaining to dorsolateral prefrontal function, in this population. Study 2 examined emotion recognition and the attribution of aggression in emotional face expressions, abilities of which are attributed to orbitofrontal functioning. Lastly, an investigation into additional orbitofrontal functions was conducted; namely inhibition, response reversal and decision-making capacities.

8.1 Overview of findings

8.1.1 Executive functioning

The first study assessed performance of impulsive-aggressive, premeditated-aggressive and control participants on a range of executive functioning tasks, including measures of cognitive flexibility, planning, problem-solving, verbal fluency, and inhibition. All measures were purported measures of dorsolateral prefrontal functioning. In line with hypotheses, the impulsive-aggressive individuals demonstrated impairment on all aforementioned measures.

The results from Study 1 were congruent with and built on past research demonstrating a link between executive deficits and the propensity for a reactive,

impulsive subtype of aggressive responding (e.g., Stanford et al., 1997). However, while previous research on impulsive-aggression has reported prefrontal deficits more broadly, these findings implicate the dorsolateral region more specifically in mediating this aggressive subtype. In contrast, no impairment was found in the premeditated-aggressive group. This finding was expected given the controlled and purposeful action which characterises such individuals' aggressive display. Taken together, these results suggest distinct roles of the dorsolateral prefrontal cortex in the expression of impulsive- and premeditated-aggression.

Executive dysfunction, through impairment in the dorsolateral prefrontal cortex, may contribute to a propensity for impulsive-aggression in a number of ways. Aggression is a primal social response option and provides a response option in social environments in which individuals are faced with multiple complex cues. As Hoaken et al. (2003) suggested, it may be that individuals with poor executive functioning who social information processing deficits are unable to cope with competing response options in interpersonal interactions. In this way, they fail to access more socially appropriate responses and consequently react aggressively in provocative situations.

Fuster (1997) proposed that the prefrontal cortex is essential in tasks that require the temporal integration of information. If an individual is overwhelmed by information in the social environment, such processes may not work adequately. This can subsequently lead to behaviour which is not appropriate for the situation. Thus, prefrontal function may lead to impaired regulation of social behaviour through an interruption in the synthesis of external and internal cues underlying the regulation of complex social behaviour. Behaviour consequently becomes governed more by impulsivity, the current focus of attention, or salient social cues, rather than by social

rules or plans. In this way, aggression would be more likely to be the response in those situations where the salient cues are provocation or hostility in others (Lau et al., 1995).

This suggestion is in parallel to Kane and Engle's (2002) position on executive functioning. They propose that a primary function of the dorsolateral prefrontal cortex is to maintain representations in memory when faced with interference. Such representations may reflect the individual's plans, goals, or task-relevant stimuli in the environment. According to Kane and Engle, failures to maintain such goal states result in incorrect information and response tendencies being retrieved. In the case of aggressive responding, impulsive-aggressive individual's executive deficit may cause an inability to follow preconceived plans and goals, resulting in a tendency to rely on reflexive aggressive responding. The probability of such interference occurring increases significantly in social interactions in which there are multiple stimuli requiring response coupled with heightened anger and frustration.

Antisocial traits as outcome behaviours can be arrived at by a variety of pathways. Morgan and Lilienfeld (2000) suggest that some aetiologies of antisocial behaviour are related to impaired executive functioning, while APD is not. However, their finding that executive skill deficits are associated with such heterogeneous classifications as „criminality' or „delinquency' does not explain which aetiologies of antisocial behaviour are linked with these cognitive deficits. The current study attempted to clarify this matter. The finding of significant differences between impulsive- and premeditated-aggressive individuals supports the idea that executive dysfunction is differentially present in antisocial individuals depending on the aetiology of those antisocial traits and behaviours. Further research with other narrowly defined antisocial groups is needed to more fully map these differences.

8.1.2 Emotion recognition and aggression attribution

Study 2 explored the interpretation of emotional facial expressions and possible hostile attributional biases in impulsive- and premeditated-aggressive individuals. Contrary to hypotheses, while the impulsive-aggressive group were not impaired on either measure, the premeditated-aggressive group attributed greater levels of aggression to neutral expressions, suggesting a tendency to a hostile attribution bias within this population.

Of interest is the relationship between the ability to accurately interpret facial expressions and success on measures of executive function. There is evidence, especially within the schizophrenia research literature, to suggest that executive cognitive capacities and emotional processing may be related (e.g., Kee, Kern & Green, 1998; Kohler, Bilker, Hagendoorn, Gur & Gur, 2000). Whether these associations between emotion recognition and executive functioning are an exclusive hallmark of schizophrenia is unclear, however it is possible that these deficiencies exist in others, and may play a role in other types of poorly regulated behaviour. Hoaken et al. (2007) tested this hypothesis, finding that violent offenders performed significantly poorer on measures of executive function and facial affect recognition than non-violent offenders and controls. This relationship was not found in the current study, finding distinct cognitive processes between individuals who are characteristically impulsive-aggressive and those who are characteristically premeditated-aggressive. Thus, while the present sample display dysregulated behaviour, a combination of executive and emotion recognition deficits do not contribute to the presentation of these behaviours. Rather, such cognitive processes represent individual and separate deficits in mediating the propensity for aggressive behaviour in the identified aggressive population.

8.1.3 Inhibition, response reversal, and decision-making

Study 3 investigated whether impulsive- or premeditated-aggressive individuals present with inhibition, response reversal, and/or decision-making dysfunction. The hypotheses that the impulsive-aggressive group would demonstrate deficits on measures of inhibition and decision-making were not supported. Similarly, contrary to hypotheses, neither impulsive-aggressive nor premeditated-aggressive groups were impaired on a measure of response reversal.

Previous research has demonstrated inhibition, response reversal, and decision-making deficits in individuals with APD (e.g., Dinn & Harris, 2000). Such impairments have also been found in individuals with lesions to the orbitofrontal cortex who present with disinhibition, impulsivity, irritability, and antisocial behaviour (Cummings, 1995). Similarly, psychopaths, who present with marked premeditated aggression, have been found to have response reversal deficits pertaining to the orbitofrontal cortex (Mitchell et al., 2002).

Dinn and Harris (2000) suggested that such impairment may reflect an inability to effectively process feedback information regarding reward and punishment (i.e., the inability to successfully employ punishment cues to guide behaviour), an ability mediated by the orbitofrontal cortex (Rolls, 2002). It may then be that individuals who do not meet the criteria for APD (e.g., subclinical populations) do not have impairment in this ability and thus do not engage in such criminal behaviour. Such individuals are able to effectively process feedback information regarding reward and punishment and thus employ such cues to guide behaviour. In this way, while they have some similar characteristics, aggressive individuals such as those identified in the current study do not engage in behaviour as severe as to meet criteria for APD.

Thus, although the findings outlined above have been taken to suggest a relationship between orbitofrontal impairment and aggressive behaviour, the analysis of a more explicitly defined aggressive population does not support this relationship. These results suggest that it may be the cumulative impact of impairment in both the dorsolateral prefrontal cortex and orbitofrontal cortex which modulates the expression of clinically significant antisocial behaviour. In contrast, individuals who present with subclinical levels of aggression have impairment solely in the dorsolateral region of the prefrontal cortex.

8.2 Theoretical implications

The results suggest that differences in prefrontal functioning between impulsive- and premeditated-aggressive individuals might contribute to the variability in aggressive responding between the groups. Evidence from animal and human studies suggests that multiple brain systems exert control over aggressive behaviour, and the type of aggression that occurs may depend on the brain system that is damaged (Weiger & Bear, 1988). The current research provides further evidence for this hypothesis.

Impulsive-aggressive individuals and patients with orbitofrontal lesions appear to share similar locus of neuroanatomical disruption, but the extent and nature of the pathology is presumably quite different. While it was hypothesised that impulsive-aggressive individuals would present with impairment on measures of orbitofrontal functioning, this was not found. Rather, impulsive-aggressive individuals showed deficits on several putative dorsolateral prefrontal cortex markers, compared with premeditated-aggressive individuals and controls. It thus appears that impulsive-aggression as identified in the present study is mediated by different regions in

comparison to clinically significant levels of aggression. Thus, one possible explanation for the differences is that impulsive-aggressive individuals have a milder form of brain dysfunction.

These results, in concert, allow for some tentative theorising on the cognitive and social-perceptual concomitants of impulsive- and premeditated-aggression. It may be that impulsive-aggressive individuals, through executive function impairments, lack the cognitive flexibility to alter their behaviour and the ability to access and utilise more adaptive response options. In contrast, those individuals characterised by high levels of premeditated-aggression are more likely to inaccurately interpret subtle social cues. That is, in ambiguous social interactions, premeditated-aggressive individuals are more likely to attribute greater levels of hostility in others. This way of interpreting social cues may increase hostility and negative arousal, which may result in a heightened aggressive response towards a situation that was initially non-threatening. By engaging in an aggressive response, the premeditated-aggressive individual could create a potentially aggressive interaction. In line with social learning theories, if such aggressive responding leads to goal attainment, such individuals learn that aggressive responding is a viable means of achieving one's objective.

The neuropsychological evidence provided by the present study, in the context of previous findings, provides strong support for the functional specialisation of the prefrontal cortex. Indeed, it suggests specialisation of the prefrontal cortex for separable systems of social cognition. One system, mediated by the dorsolateral prefrontal cortex, allows the successful execution of executive functions. The second system, incorporating the orbitofrontal cortex, responds to facial expressions of emotion and the expectation of others' anger. While such hypotheses are tentative, it does call for further research to investigate the complex relationships between

executive functioning, arousal, and social perception and the propensity for aggressive behaviour.

8.2.1 Inhibition

The two measures of inhibition, the Stroop and Stop Signal tasks seem to draw into question the predominant disinhibition explanation of the executive functioning-aggression relationship. On the Stroop task, it was expected that the impulsive-aggressive group would make more errors on the interference condition, indicating an inability to inhibit responding. This hypothesis was partially supported, with the impulsive-aggressive group making more errors than the control group, but not differing significantly from the premeditated-aggressive group. On the second measure of inhibition, the Stop Signal Task, impulsive-aggressive individuals were expected to make more errors and have shorter response times than premeditated-aggressive individuals and controls. However, no significant group differences were found. Furthermore, while participants in the impulsive-aggressive group failed to inhibit responses on the Stroop task, the reaction-time data for these participants are contrary to the expectancy that rapid action, and an absence of adequate forethought, characterises impulsive-aggression. These two putative measures of impulsivity thus seem to lend contradictory support for the inhibition hypothesis.

Given the lack of definitional agreement in the clinical literature regarding impulsivity, investigating the link between aggression and impulsivity is complex (Parker, Bagby & Webster, 1993). There exist numerous self-report measures of impulsivity, all of which conceptualise impulsivity slightly differently (see Parker & Bagby, 1997 for review). Furthermore, correlational research has identified inconsistent relationships between them (Luengo, Carrillo-de-la-Pena & Otero, 1991).

There are also several behavioural measures of impulsivity which do not correlate (Gorlyn et al., 2005). These behavioural measures are based on the theories that impulsive individuals have a tendency to make quick decisions and act without thinking, and that they tend to overestimate the amount of time that has passed. Of importance to the current study is that correlational studies have found no relationship between self-report and behavioural measures of impulsivity (Dolan & Fullam, 2004; Helmers, Young & Pihl, 1995; Malle & Neubauer, 1991). In this way, the findings in the present study may simply represent a definitional discrepancy in the impulsivity construct. That is, although demonstrating impulsive-aggressive behaviour, this inhibition deficit may be context-specific. That is, in situations of heightened arousal and complex social stimuli, impulsive-aggressive individuals are unable to inhibit such responding. In contrast, in experimental situations in which the individual is not experiencing anger they are able to inhibit responding.

The research question then becomes whether cognitive dysfunction becomes prominent in the presence of heightened emotion, at least in subjects with high levels of impulsive-aggression. It is known that stress (e.g., emotional distress) correlates with lower performance in cognitive tasks (Eysenck & Calvo, 1992; Yee & Vaughan, 1996). Thus, it may be that in states of high emotional reactivity, limited mental resources might trigger cognitive impairment. Therefore, future studies should not examine cognitive processes as if they were independent of emotional states.

In line with this theory, evidence suggests that the relation between difficulties with frontal lobe functioning and aggressive behaviour may be contingent on the presence of inhibition cues (Giancola, 2000). As Lau et al. (1995) proposed, difficulties on measures of executive functioning should not necessarily contribute to increased levels of aggression unless individuals are in situations in which "the salient

cue is provocation and where peripheral or less contingent cues that inhibit aggression are lessened" (p. 150). As suggested above, this argument implies that the effect of executive functioning difficulties on aggression is context dependent. In such contexts there must be cues that generally inhibit aggression which are ignored by impulsive-aggressive individuals. In contrast, those who do not have executive deficits are able to process social cues adequately and thus respond in a more socially appropriate manner.

8.2.2 Premeditated-aggression

The premeditated-aggressive individuals did not demonstrate impairment on measures of dorsolateral functioning compared to controls. This is consistent with the view that such individuals have relatively intact ability to plan and regulate their aggressive behaviour in order to achieve desired goals. Premeditated-aggressive individuals did however attribute greater levels of hostility to neutral faces of emotion than did the impulsive-aggressive and control groups. This would lead to a greater experience of negative affect from others and may contribute to the greater incidence of aggressive responding toward others.

However, while a hostile attribution bias may contribute to the likelihood of premeditated-aggression, it is unlikely that this represents the only cognitive mediator in increasing the susceptibility to display such aggressive behaviour. Premeditated-aggression can be considered as instrumental, and a form of controlled behaviour. Goal directed behaviours are performed in expectation of receiving the desired reward and if they are not punished. Moral socialisation enables an individual to learn that aggressive behaviour is not the ethical manner in which to obtain his/her desired goal.

In this way, it may be presumed that socialisation has not been achieved in those displaying high levels of premeditated-aggression (Blair, 2007).

As Blair (2007) outlined, there are two capacities necessary for successful socialisation. Firstly, the individual must find the distress of others aversive. Such distress may be manifested in expressions of fear and sadness which serve as social reinforcers (Blair, 2003b). Secondly, the individual must be capable of performing stimulus-reinforcement learning. Psychopaths show impairment in both of these capacities (Blair, 2003a; Budhani et al., 2006), and it is thus proposed that psychopaths do not learn to avoid using aggression to achieve their goals. This is due to a relative lack of concern to the victim's distress (i.e., punishment) and impairment in learning the association between this punishment and the representation of the action that caused the distress.

The ability to respond to distress cues and to achieve stimulus-reinforcement learning are both related to the functioning of the amygdala. Neuroimaging research demonstrates that while the amygdala may respond to expressions generally, it is most responsive to fearful expressions (Adolphs, 2002b). Similarly, neuroimaging research has also implicated the amygdala in stimulus-reinforcement learning in both aversive conditioning and passive avoidance learning (Kosson et al., 2006; LaBar, Gatenby, Gore, LeDoux & Phelps, 1998). These data have prompted the suggestion that psychopathy relates to dysfunction in the amydala's role in expression processing and stimulus-reinforcement learning (Blair, 2003a; Blair, Peschardt, Budhani, Mitchell & Pine, 2006)

While psychopaths show impairment in both of these capacities, the premeditated-aggressive individuals in the current study have not. The level of premeditated-aggression identified in the present study thus appears to be the result of

behavioural choices; the individual had other behaviours available to meet his or her goals or could have chosen different goals. It is suggested that premeditated-aggressive individuals choose these behaviours not because of deficient decision-making; but because the behaviour is believed to meet their identified goal.

8.3 Clinical Implications

Given the putative underlying mechanisms of impulsive- and premeditated-aggression, there are some potential implications for intervention. Clients with aggressive traits often have significant and chronic problems with poor anger control, lack of empathy and/or remorse, irresponsibility, and impulsivity, all of which can impede successful treatment. The current findings indicate that individuals whose aggressive behaviour is predominantly premeditated in nature do not have difficulties with executive behaviours that require planning, problem-solving, purposeful action, and abstract reasoning in comparison to those whose aggressive behaviour is impulsive in nature. Thus, it would seem more valid for clinicians to conceptualise the client's behaviour as either predominantly impulsive or predominantly premeditated in nature in order to determine the appropriate treatment program. In turn, this may help clinicians channel their therapeutic efforts to these potentially more malleable domains in order to effect behavioural change.

The current results suggest an association between executive dysfunction and impulsive-aggression. Programs that would be most effective with such a population should thus aim to improve or restore cognitive ability. Such programs should encompass cognitive training in executive functions such as problem-solving, planning, selective attention, abstract reasoning, judgement, inhibition, learning from

experience and cognitive self-monitoring of behaviour during goal-directed activities (Paschall & Fishbein, 2002).

Deficits in cognitive flexibility could contribute to the expression of impulsive-aggression through an inability to effectively find an alternative solution to a confrontational situation. If cognitive flexibility is correlated with poor problem-solving and deficiencies in generating alternative non-aggressive responses, enhancing the ability to think flexibly should lead to a decrease in aggressive responses. For example, Ross, Fabiano and Ewles (1988) found that engaging offenders in alternative ways of thinking about problems or to improve their creativity in problem-solving was the most useful in rehabilitation. Further to this, Slaby and Guerra (1990) also found that by encouraging the discovery of alternative responses, aggressive responses could be reduced.

Techniques for inhibiting inappropriate behaviours could utilise associations between cues and consequences, which serve to slow down or stop the behaviour. The emphasis should be on drawing the individual's attention to the behaviour and to its consequences, and in this way the individual becomes aware of their behaviour at specific points in time and can make the identified adjustments. Such adjustments can then be either rewarded or punished in order to highlight the consequences of the behaviour (Paschall & Fishbein, 2002).

Research has demonstrated that it also possible to employ the same tools used to assess executive cognitive skills (i.e., neuropsychological tests) to strengthen such these abilities (see Giancola, 2000 for review). Repeat performances of these instruments appear to improve executive abilities by teaching techniques to delay gratification, inhibit prepotent responses, shift strategies to produce more advantageous consequences, and reinforce mood regulation and behavioural controls.

Computerised versions of these assessment instruments are also able to be programmed with a hierarchy of different levels so that as executive cognitive capacity increases, the individual could attempt more demanding versions. One particular example of the success of cognitive rehabilitation techniques come from New South Wales where individuals with aggressive, disinhibited, and socially inappropriate behaviours were treated. Using tokens to reward self-regulated behaviours and withholding the tokens at scheduled times when behaviours were inappropriate resulted in a substantial reduction of violent episodes (Manchester, Hodgkinson & Casey, 1997).

8.4 Limitations

There are several limitations to the current research. The use of university students as a sample, while providing some evidence of prefrontal dysfunction within a subclinical population, restricts external validity. Similar research using a wider range of participants from the general community is needed to overcome this issue.

This study used traditional neuropsychological tests which are believed, based on lesion and neuroimaging studies, to reflect brain dysfunction in specific areas. However, it is acknowledged that such measures cannot be attributed specifically to one region. Rather, the successful completion of such tasks depends on a wide range of brain regions which are intrinsically associated with each other. Further research using new functional and structural neuroimaging techniques will contribute to a greater understanding of the neural underpinnings of cognitive abilities and consequently to the knowledge of brain-behaviour relationships. Furthermore, while this research has investigated cognitive abilities thought to be associated with aspects of the prefrontal cortex, it is difficult to make causal commentaries about cognition

and structure in the absence of specific neuroimaging data. In the absence of these data, future studies would be strengthened by the assessment of specifically aggressive individuals using neuroimaging techniques.

8.5 Conclusion

In conclusion, this study provides some preliminary evidence for separable roles of regions of the prefrontal cortex in impulsive- and premeditated-aggression. The results suggest that impulsive-aggressive individuals demonstrate impairment across a wide range of executive domains, including verbal fluency, cognitive flexibility, planning, problem-solving and inhibition suggesting potential dorsolateral prefrontal dysfunction in impulsive-aggressive individuals. This is consistent with data demonstrating significant correlations between neuropsychological measures of frontal lobe function and impulsive-aggression in incarcerated samples and head-injured samples. In contrast, premeditated-aggressive individuals, while not presenting with executive impairment, attribute greater levels of hostility to neutral faces of emotion, abilities of which are predominantly mediated by the orbitofrontal region of the prefrontal cortex.

These findings suggest distinct cognitive processes between impulsive-aggressive individuals and those whose aggressive behaviour is premeditated in nature. Furthermore, the results have helped to delineate the hypothesised relationship between prefrontal functioning and antisocial behaviour. While previous research has suggested a relationship between orbitofrontal dysfunction and impulsive antisocial behaviour, the current results indicate that such relationship does not pertain to aggressive behaviour more specifically. Rather, impulsive-aggression in a subclinical population appears to be mediated by primarily the dorsolateral region of the

prefrontal cortex, while premeditated-aggression may be related to dysfunction in the orbitofrontal cortex through its involvement in emotion recognition.

While such theories are preliminary, it is hoped that knowledge gained from this study will encourage further research into the cognitive processes of aggressive individuals in a non-clinical population. Further investigations will enhance the understanding of the cognitive and social-perceptual underpinnings of aggression and may be used to better inform contemporary intervention strategies for aggressive behaviour.

In light of these findings, it is important to note that such conclusions regarding the role of the prefrontal cortex in aggression are simplistic. Such results do not suggest that the dorsolateral and orbitofrontal cortex areas are the only cortical areas involved in the propensity for impulsive- and premeditated-aggression. These specific brain regions are richly connected with other neural regions, cortical and subcortical. Thus it may be the modulation between these identified brain regions that increases the susceptibility to aggressive responding.

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Appendices

Appendix A: HREC Approval Letter

Appendix B: Information sheet for Study 1

Appendix C: Consent form for Study 1

Appendix D: Facial stimuli from Ekman and Friesen's (1976) collection used for the emotion recognition task.

Appendix E: Facial stimuli from Ekman and Friesen's (1976) collection used for the aggression rating task.

Appendix F: Information sheet for Study 2 and Study 3

Appendix G: Consent form for Study 2 and Study 3

Appendix A: HREC Approval Letter

Private Bag 01 Hobart
Tasmania 7001 Australia
Telephone (03) 6226 2764
Facsimile (03) 6226 7148
Marilyn.Knott@utas.edu.au
http://www.research.utas.edu.au//index.htm

MEMORANDUM

HUMAN RESEARCH ETHICS COMMITTEE (TASMANIA) NETWORK

UTAS

FULL COMMITTEE APPLICATION APPROVAL

25 July 2007

Dr Frances Martin
Psychology
Private Bag 30
Hobart

Ethics reference: H9497

'The role of the Prefrontal Cortex in the expression of impulsive and premeditated aggression'.

PhD candidate: Sarah Haberle

Dear Dr Martin

The Tasmania Social Sciences HREC Ethics Committee approved the above project on 25 July 2007.

All committees operating under the Human Research Ethics Committee (Tasmania) Network are registered and required to comply with the *National Statement on the Ethical Conduct in Research involving Humans 1999* (NHMRC guidelines).

Therefore, the Chief Investigator's responsibility is to ensure that:

1) All researchers listed on the application comply with HREC approved application.
2) Modifications to the application do not proceed until approval is obtained in writing from the HREC.
3) The confidentiality and anonymity of all research subjects is maintained at all times, except as required by law.
4) Clause 2.37 of the *National Statement* states:
   An HREC shall, as a condition of approval of each protocol, require that researchers immediately report anything which might warrant review of ethical approval of the protocol, including:
   a) *Serious or unexpected adverse effects on participants;*
   b) *Proposed changes in the application; and*
   c) *Unforeseen events that might affect continued ethical acceptability of the project.*

   The report must be lodged within 24 hours of the event to the Ethics Executive Officer who will report to the Chairs.

5) All participants must be provided with the current Information Sheet and Consent form as approved by the Ethics Committee.

6) The Committee is notified if any investigators are added to, or cease involvement with, the project.

7) This study has approval for four years contingent upon annual review. An *Annual Report* is to be provided on the anniversary date of your approval. Your first report is due [12 months from 'Ethics Committee Approval' date]. You will be sent a courtesy reminder by email closer to this due date.
Clause 2.35 of the National Statement states:
As a minimum an HREC must require at regular periods, at least annually, reports from principal researchers on matters including:
   a) *Progress to date or outcome in case of completed research;*
   b) *Maintenance and security of records;*
   c) *Compliance with the approved protocol, and*
   d) *Compliance with any conditions of approval.*

8) A *Final Report* and a copy of the published material, either in full or abstract, must be provided at the end of project.

Yours sincerely

M. Knott

Ethics Executive Officer

for

Appendix B: Information Sheet for Study 1

Research Project on Personality Differences
Information Sheet for Participants

This research is about personality and behaviour and will investigate the ways in which people with difference personality styles differ and how these differences impact on information processing. The research is being conducted by Sarah Haberle, a PhD student, under the supervision of Dr Frances Martin and Dr Clive Skilbeck. This research is primarily interested in group data. Individual results will not be identifiable at any stage of the data analysis and reporting.

Based on your responses to a questionnaire completed in your first year psychology prac class, you have been invited to take part in further stages of this research project. Participation in this project will involve some very simple procedures. If you have any questions about the nature of the study, please feel free to ask at any stage. Participation is completely voluntary, and you may withdraw at any time without penalty. It is up to you whether or not you wish to take part. If you choose to participate, there are several activities that you will be asked to complete. These are detailed below.

Firstly, you will be asked to complete a pattern recognition task, which consists of viewing a series of cards with a particular pattern printed on them. You are required to guess the pattern presented on the following card. Accuracy will be recorded on this task.

Secondly you will be asked to complete a simple verbal task which requires you to generate as many words as possible in one minute beginning with a specific letter. There will be three trials in this task and your performance will be indexed by the number of words produced.

The third task comprises of five separate conditions which involves joining a sequence of numbers and/or digits in a specific order as quickly as possible.

The fourth task is a computerised game which requires you to move five rings which differ in circumference from one rod to another rod according to certain rules. Performance is indexed by the number of moves taken to produce the stacking configuration.

The fifth task is also computerised and involves watching for and responding to certain colours and words. This is a very simple task and you should be able to complete it even if you do not have any previous experience with computers or video games. Reaction time and accuracy will be recorded on this task.

The sixth task requires you to provide oral definitions for words which the experimenter states.

Finally, the seventh task is composed of two conditions. On both subtests, the experimenter will read aloud a series of numbers. For the first condition, you are required to repeat the number sequence in the same order as presented. For the second condition, you are required to repeat the number sequence in the reverse order.

Following the completion of these tasks, you will be asked to complete two questionnaires relating to different aspects of personality and behaviour.

Completion of these tasks and questionnaires will take approximately one hour. You will receive one hour course credit upon the completion of all tasks and questionnaires.

Your personal data which was obtained from the screening questionnaire will be kept in a coded form and the information linking these codes to individuals will be securely stored in a locked filing cabinet. Personal contact information will be destroyed after the completion of data collection. The information obtained from testing will be stored in a locked filing cabinet for a minimum of five years, accessible only by the investigators identified below. Results will be presented and publicised, however no results will involve identification of individual participants in any way, as this research is purely interested in group data. Please note that subsequent to the project you will be given a full debriefing on the project.

Your participation in this project is entirely voluntary, and evidenced by signing a consent form. You can, in any case, withdraw from the project at any time during your participation.

This research project has the approval of the Human Research Ethics Committee (Tas) Network and the School of Psychology. If you have any concerns of an ethical nature or complaints about the manner in which the project is conducted, please contact the Executive Officer of the Network, Ms Nadia Mahjouri (6226 7479). Alternatively, you may prefer to discuss any concerns confidentially with a University Student Counsellor, who are located on the top floor of the TUU building and can be contacted on 62 262697.

This project will be conducted by Sarah Haberle as a component of her PhD in Clinical Psychology, and is to be supervised by Dr Frances Martin and Dr Clive Skilbeck who lecture within the School of Psychology. Please keep this information sheet, and if you have further queries please call Sarah Haberle (62262260). A summary of the results of the project will be available on the School of Psychology website at year end.

Thank you.

Sarah Haberle (PhD student)

Appendix C: Consent form for Study 1

Research Project on Personality Differences

Statement of Informed Consent

Participants: Please read, sign and date this form.

I have read and understood the information sheet for this research. The nature and possible effects of the research, and the activities that I will be involved in have been explained to me. I understand that my participation in this project will involve completing a series of different tasks, involving computer tasks, verbal tasks, and pattern recognition tasks. I understand that these tasks should not be distressing in any way, that the tasks will be explained in a way that I can understand, and that anonymity will be assured at all times. Any questions that I have asked have been answered to my satisfaction, and I am aware that I may contact the researchers if I have further queries or concerns. I also understand that I can withdraw from the research at any time without prejudice, and that I will have access to a copy of the research report on its completion if I so wish.

I, ……..(your name), hereby consent to take part in this research and agree that data gathered for the research may be published, provided that my identity is not revealed.

.....

(Signature of Participant)

Date __/__/20

To be filled in by researcher at time of assessment:

I have explained this research to .....…….. (participant's name). I believe the consent is informed and that he/she understands the implications of participation and that he/she may withdraw at any time without prejudice.

.....

(Researcher's Signature)

Date __/__/20

Appendix D: Facial stimuli from Ekman and Friesen's (1976) collection used for the emotion recognition task

Aggressive Faces

EM5-14; JJ3-12; MF2-7; MO2-11; NR2-7; PE2-21; SW4-9; WF3-1

Disgusted Face

EM4-17' JJ3-20; MF2-13; MO2-18; NR3-29; PE4-5; SW1-30; WF3-11

Frightened Faces

EM5-21; JJ5-13; MF1-26; MO1-21; NR1-19; PE3-21; SW2-30; WF3-16

Happy Faces

EM4-7; JJ4-7; MF1-6; MO1-4; NR1-6; PE2-12; SW3-9; WF2-12

Surprised Faces

EM2-11; JJ4-13; MF1-9; MO1-14; NR1-14; PE6-2; SW1-16; WF2-16

Sad Faces

EM4-24; JJ5-5; MF1-30; MO1-30; NR2-15; PE2-31; SW2-16; WF3-28

Neutral Faces

EM2-4; JJ3-4; MF1-2; MO1-5; NR1-3; PE2-4; SW3-3; WF2-5

Appendix E: Facial stimuli from Ekman and Friesen's (1976) collection used for the aggression rating task

Aggressive Faces

EM5-14; C2-12; CS2-8; JJ3-12; MF2-7; MO2-11; NR2-7; PE2-21; SW4-9; WF3-1

Disgusted Face

EM4-17; C1-4; CS2-25; JJ3-20; MF2-13; MO2-18; NR3-29; PE4-5; SW1-30; WF3-11

Frightened Faces

EM5-21; C1-23; GS1-25; JJ5-13; MF1-26; MO1-21; NR1-19; PE3-21; SW2-30; WF3-16

Happy Faces

EM4-7; C2-18; GS1-8; JJ4-7; MF1-6; MO1-4; NR1-6; PE2-12; SW3-9; WF2-12

Neutral Faces

EM2-4; C2-3; GS1-4; JJ3-4; MF1-2; MO1-5; NR1-3; PE2-4; SW3-3; WF2-5

Appendix F: Information sheet for Study 2 and Study 3

Research Project on Personality Differences

Information Sheet for Participants

This research is about personality and behaviour and will investigate the ways in which people with difference personality styles differ and how these differences impact on processing of social information. The research is being conducted by Sarah Haberle, a PhD student, under the supervision of Dr Frances Martin and Dr Raimondo Bruno. This research is primarily interested in group data. Individual results will not be identifiable at any stage of the data analysis and reporting.

Based on your responses to a questionnaire completed in your first year psychology prac class, you have been invited to take part in further stages of this research project. Participation in this project will involve some very simple procedures. If you have any questions about the nature of the study, please feel free to ask at any stage. Participation is completely voluntary, and you may withdraw at any time without penalty. It is up to you whether or not you wish to take part.

If you choose to participate, there are several activities that you will be asked to complete. These are detailed below.

You will be asked to sit in front of a computer screen and complete a simple computerised game involving watching for and responding to target items on the screen. This is a very simple task and you should be able to complete it even if you do not have any previous experience with computers or video games. Reaction time and accuracy will be recorded on this task.

Next, you will be asked to complete a mock gambling task. This task involves selecting cards off one of four decks which may yield either a high or low reward or high or low punishment. The measure for this task is the amount of money you have at the end of the task.

The third task requires you to discriminate and respond differently to a series of letters and numbers shown on the screen using various keys on the keyboard. Reaction time and accuracy will be recorded on this task.

The fourth task requires you to view a series of faces displayed on the screen. After viewing each face, you will be asked to rate each face with respect to provided adjectives.

For the final task you will be asked to view a series of emotional faces and state which emotion the face is displaying.

Following the completion of these tasks, you will be asked to complete three questionnaires relating to different aspects of personality and behaviour.

Completion of these tasks and questionnaires will take approximately two hours. You will receive two hours course credit upon the completion of all tasks and questionnaires.

Your personal data which was obtained from the screening questionnaire will be kept in a coded form and the information linking these codes to individuals will be securely stored in a locked filing cabinet. Personal contact information will be destroyed after the completion of data collection. The information obtained from testing will be stored in a locked filing cabinet for a minimum of five years, accessible only by the investigators identified below. Results will be presented and publicised, however no results will involve identification of individual participants in any way, as this research is purely interested in group data. Please note that subsequent to the project you will be given a full debriefing on the project.

Your participation in this project is entirely voluntary, and evidenced by signing a consent form. You can, in any case, withdraw from the project at any time during your participation.

This research project has the approval of the Human Research Ethics Committee (Tas) Network and the School of Psychology. Should you have any concerns, questions or complaints with regard to the ethical conduct of this research, please contact the Executive Officer of the Human Research Ethics (Tasmania) Network, on 6226 7479 or human.ethics@utas.edu.au. Alternatively, you may prefer to discuss any concerns confidentially with a University Student Counsellor, who are located on the top floor of the TUU building and can be contacted on 62 262697.

This project will be conducted by Sarah Haberle as a component of her PhD in Clinical Psychology, and is to be supervised by Dr Frances Martin and Dr Raimondo Bruno who lecture within the School of Psychology. Please keep this information sheet, and if you have further queries please call Sarah Haberle (skh@utas.edu.au, 6226 2260), Dr Frances Martin (F.Martin@utas.edu.au, 6226 2262) or Dr Raimondo Bruno (Raimondo.Bruno@utas.edu.au, 6226 2240). A summary of the results of the project will be available on the School of Psychology website at year end.

Thank you.

Frances Martin (Chief Investigator) Sarah Haberle (PhD Student)

Appendix G: Consent form for Study 2 and Study 3

Research Project on Personality Differences
Statement of Informed Consent

Participants: Please read, sign and date this form.

I have read and understood the information sheet for this research. The nature and possible effects of the research, and the activities that I will be involved in have been explained to me. I understand that my participation in this project will involve completing a series of different tasks, involving computer tasks, face rating tasks and questionnaires. I understand that these tasks should not be distressing in any way, that the tasks will be explained in a way that I can understand, and that anonymity will be assured at all times. Any questions that I have asked have been answered to my satisfaction, and I am aware that I may contact the researchers if I have further queries or concerns. I also understand that I can withdraw from the research at any time without prejudice, and that I will have access to a copy of the research report on its completion if I so wish.

I, ……..(your name), hereby consent to take part in this research and agree that data gathered for the research may be published, provided that my identity is not revealed.

.....

(Signature of Participant)

Date __/__/2009

To be filled in by researcher at time of assessment:

I have explained this research to .....……..
(participant's name). I believe the consent is informed and that he/she understands the implications of participation and that he/she may withdraw at any time without prejudice.

.....

(Researcher's Signature)

Date __/__/2009